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MEDICAL OPHTHALMOLOGY
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AN INTERNATIONAL SYSTEM
OF
OPHTHALMIC PRACTICE

Edited by WALTER L. PYLE, A. M., M. D.
Member of the American Ophthalmological Society,
Formerly Ophthalmologist to the Mt. Sinai
Hospital, Philadelphia, Assistant Surgeon
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MEDICAL OPHTHALMOLOGY

BY
ARNOLD KNAPP, M. D.
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MEMORIAL EYE HOSPITAL.

WITH THIRTY-TWO ILLUSTRATIONS

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PREFACE

Hughlings Jackson concluded his presidential address at the annual meeting of the Ophthalmological Society of the United Kingdom in 1890 by adopting the following words of Dr. James Anderson (Some Ocular and Nervous Affections in Diabetes and Allied Conditions—*Ophthalmic Review*, 1889): "It seems to me the best and most hopeful feature of ophthalmology is that it has relations, closer or more remote, with every branch of medicine and surgery; indeed, with almost every branch of science." These relations are to-day closer than ever and are important not only for the eye-specialist but even for the general practitioner.

The aim of this book has been to give the essential of what is known of these relations at the present day. In doing this, I have freely drawn from the authoritative monographs in the Graefe-Saemisch-Hess *Handbuch* II Ed., particularly from the parts written by Uhthoff, Groenouw and Leber. I have moreover received great help from Lewandowsky, *Handbuch der Neurologie*, the chapters of Henschen in particular, Wilbrand und Sänger, *Neurologie des Auges*, de Lapersonne et Cantonnet, *Neurologie Oculaire* and from the *Transactions* of the Ophthalmological Society of the United Kingdom. The indebtedness is furthermore acknowledged by appropriate footnotes in which the names of the authors consulted are given.

It has seemed desirable to introduce the subject matter with an illustrated description of ophthalmological anatomy, especially of the anatomy of the nervous system connected with sight. In this part I was fortunate enough to enlist the coöperation of Professor Otto Marburg of Vienna and it is with pleasure that I take this opportunity of expressing my deep gratitude.

ARNOLD KNAPP.

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MEDICAL OPHTHALMOLOGY

I. INTRODUCTORY ANATOMY AND PHYSIOLOGY WITH TOPOGRAPHICAL DIAGNOSIS

VISUAL PATHS

The visual paths form an incompletely crossed intercentral conducting system,[1] which, from its origin in the retina to the visual center in the cerebral cortex, constitutes an organically connected entity in which the optic fibres everywhere maintain a strictly uniform order. Each visual path consists of two neurons: (*a*) a primary neuron of the peripheric visual path from the retina to the primary optic centers in the between- and mid-brain; (*b*) a secondary neuron of the central visual path from the primary optic centers to the visual sphere in the occipital lobe of the cerebrum. The primary optic centers are interposed between the peripheric and central visual paths.

Peripheric Visual Path.—The peripheric visual path includes the retina, the optic nerve, the chiasm, and the optic tract.

RETINA.—The retina serves as the peripheric perceptive organ for the optic nerve. Our visual impressions are received by the sensory layer of the retina, the layer of rods and cones. This layer is closely connected with the underlying pigment epithelium of the choroid, which furnishes the visual purple and belongs embryologically to the retina. The rods and cones are the outer segment of cells (visual cells), whose nuclei constitute the outer granular layer. It receives its nutrition by diffusion from the choriocapillaris. The remaining layers of the retina serve for the transmission of the visual impression. The ganglion cells consist of multipolar cells which on one side are in contact with the visual cells through the intermediary of the bipolar cells which form the inner granular layer, and on the other side form a prolongation which serves as an axis cylinder in the nerve-fibre layer and later becomes an optic-nerve fibre and ends in the primary optic ganglia.

---

[1] Brodmann, p. 205. Allgemeine Chirurgie d. Gehirnkr'kh'ten Krause, Stuttgart, 1914.

A number of different neurons have been described in the visual apparatus and the grouping of the cellular bodies of the three first neurons in one membrane seems difficult to explain. According to Duval, however, there is a homology between this apparatus and other sensory nerves. The visual sensory nerve has a sensory epithelium or a neuro-epithelium (the first visual neuron), a peripheric neuron (the second) and the central neuron (the third neuron). The last has its cell body not only outside of the body but included in the sensory membrane itself, of which it forms one of the layers. The retina is therefore to be regarded as a true nervous center, because it contains a central neuron with neuroglia and because it is developed not like a separate organ, but like a prolongation of the brain. It is, in short, the equivalent of the gray matter of the brain, and is in no respect a peripheric nerve but is a true commissural nerve between two centers.

The larger blood vessels and their main sub-divisions are situated in the nerve-fibre layer, smaller retinal branches are given off externally at right angles, to end in capillary loops. The nerve-fibre layer is made up of non-medullated axis cylinders collected in bundles which converge to form the optic nerve, and leave the eyeball through the scleral foramen.

Distribution of Optic Nerve Fibres in the Retina.—The fibres run radiatingly from the papilla, up, down, and in. At the outer side they partly describe an arch, so as to encircle the macula lutea, and as the encircling fibres coming from above and below meet beyond the macula, they form a raphé in the horizontal meridian. Rönne[1] has drawn attention to a variation in visual-field defects which occurs when these two large arched retinal fibre-bundles passing over and under the macula, are unequally affected. He calls this defect "nasal step." This raphé is thus exhibited in the field as a line in the nasal horizontal meridian along which the field periphery takes a step. The macula itself is supplied by very delicate fibres which run a straight course from the temporal half of the papilla to the macula. This group of fibres is known as the papillomacular bundle and is of the greatest clinical importance.

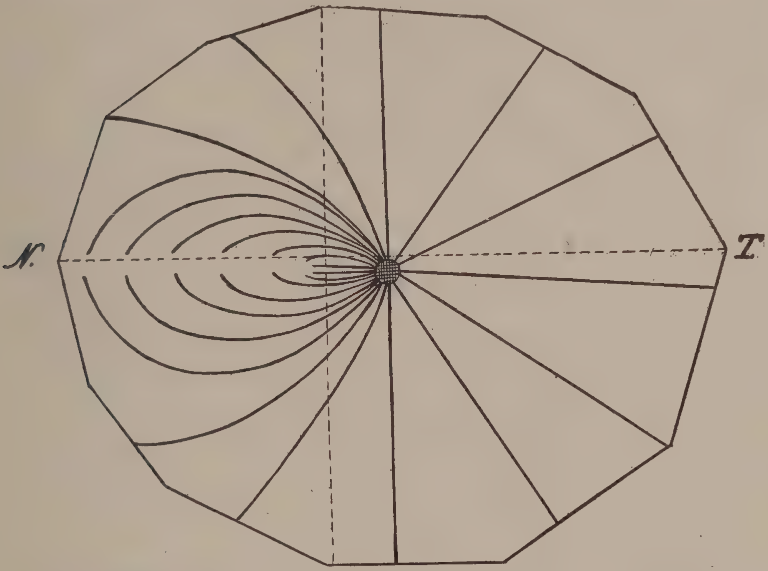

FIG. 1.—Diagram to show the course of the retinal fibres. The fibres which arch above and below the straight papillomacular fibres join to form an imaginary horizontal raphe in the nasal half of the field.

1 [illegible]

OPTIC NERVE.—The optic nerve traverses the orbital cavity imbedded in fat and important orbital structures. It is about 2 *cm* long and is described as usually making two curves. It is surrounded by three sheaths derived from the cerebral membranes, hence designated as dural, arachnoid and pial sheaths. There is a space between each two adjoining membranes continuous with the subdural and subarachnoid cerebral cavities. The dural sheath surrounds the optic nerve loosely, and at the eye ball is continued into the sclera. The inner surface of the dura is covered with a layer of continuous endothelium; this is supplied with many nerves and vessels. The former are branches of the ciliary nerves and ciliary ganglion, and extend plexus-like along the vessels which are derived from the ophthalmic artery and vein. The pain of retrobulbar neuritis is due, some believe, to an accumulation of fluid in the dural sheath.

---

[1] Graefe's Arch., Vol. 71; Arch. f. Aug., Vol. 74, p. 180.

The arachnoid is an exceedingly thin membrane directly contiguous to the dural or outer sheath. The pial sheath represents the perineurium of the nerve. From its inner surface the connective-tissue framework is given off, which forms the septal system supporting the optic nerve bundles. This sheath is the vascular layer and is richly supplied with blood vessels, derivates of the ophthalmic artery and vein. The septa carry in the nutrition (blood) to the optic nerve. Thus in inflammation the changes are, just as in the brain, located in the connective-tissue framework (interstitial neuritis) and the enclosed nerve bundles are pressed upon and atrophy. The intervaginal lymph space is situated between the pia and the arachnoid.

The optic nerve is composed of a large number of nerve fibres grouped in bundles. Each nerve fibre consists of an axis-cylinder invested in a delicate myelin sheath and surrounded by neuroglia. Within the nerve bundles there are only nerve substance and neuroglia, no blood vessels or connective tissue. This neuroglia is identical with that found in the white matter of the brain. The fibres vary in size; the thin fibres are regarded as necessary for seeing (visual fibres), while the thicker ones are pupillary fibres.

The *lymphatics* of the eyeball and optic nerve require some explanation on account of their importance. Tenon's space and the intervaginal space of the optic nerve are two lymph cavities. The former communicates with the suprachoroidal space by channels about the vortex veins. The central canal in the vitreous communicates directly with the lymph spaces of the optic nerves. This may explain the optic neuritis seen in injuries or diseases of the anterior segment of the eye. Birch-Hirschfeld[1] has endeavored to show experimentally the presence of lymph vessels in the orbit and believes that their existence, must be accepted, just as in other parts of the body. The blood vessels are probably all supplied with perivascular lymph spaces. Thus a communication exists between the lymphatic system of the orbit and important adjoining structures, such as the nose and skull, along which inflammatory processes may extend.

The *ophthalmic artery* branches off from the internal carotid artery, after the latter has emerged from the cavernous sinus and is ascending at the inner side of the anterior clinoid processes. The ophthalmic artery leaves the cranial cavity by the optic foramen inclosed in the same sheath as the optic nerve, and is situated below and to the outer side of the nerve. Within the orbit it pierces the optic sheath and proceeds forward at first on the outer side and then crosses over the optic nerve, to split up into terminal branches. The branch which interests us most is the central retinal artery. This, together with its vein, perforates the dural sheath in the lower and outer quadrant at about 10 to 15 *mm* back of the eyeball, and, surrounded by a pial mantle, gains access to the center of the nerve. The vein runs a short distance between the sheaths and enters the optic nerve somewhat nearer to the eyeball than the artery. At the point of entrance the vein is located externally and enters later after running superficially. Perhaps this makes the vein more vulnerable to pressure from accumulated cerebro-spinal fluid.[2] The vessels are surrounded by a connective-tissue mantle derived from the pial sheath, and on gaining the center of the nerve proceed straight forward to the optic papilla. In this straight course they are accompanied by two to three smaller vessels which are branches and supply the adjoining parts of the optic nerve. Vossius has shown that the central vessels, just as they reach the center of the nerve, send off large branches which pass directly back in the center of the nerve nearly to the optic foramen. The clinical importance of the definite entrance of the retinal vessels in the optic nerve is evident. Any process of the optic nerve distal to the site of entrance is accompanied by ophthalmoscopically visible changes in the retinal vessels, while a lesion further back in the optic nerve is associated with a normal eyeground.

---

[1] Birch-Hirschfeld (Graefe-Sämisch, 2d. Ed., Vol. IX). p. 262,

[2] *Wilbrand* and *Sänger*, Neurologie d. Auges, Vol. 1, p. 34.

The posterior ciliary arteries are given off at the same time as the retinal artery. They proceed forward, surrounding the optic nerve, and at the posterior pole of the eyeball perforate the sclera, to be distributed in the choroid and the ciliary processes. Pathological changes in these vessels are not so important for the eye, as they form many anastomoses in the choroid. The other branches are the lacrymal, supraorbital, ethmoidal and muscular branches.

The *veins* of the orbit are of greater pathological importance. The blood enters the cerebral circulation, except for anastomoses with the facial vein, and the pterygoid plexus (inferior orbital fissure); this explains the lack of ophthalmoscopic signs in cavernous sinus thrombosis. There are usually two ophthalmic veins in the orbit. The larger, the superior, arises at the root of the nose, communicating with the angular vein and accompanies the artery across the optic nerve. The inferior lies below the optic nerve and communicates with the pterygoid venous plexus by a branch passing through the spheno-maxillary fissure. The superior ophthalmic vein receives all the tributaries corresponding to the branches of the ophthalmic artery.

The principal ones of these are the ethmoidal veins and the venæ vorticosæ, which also empty into the inferior ophthalmic vein. The central retinal vein empties directly into the cavernous sinus or into the junction of the two ophthalmic veins before they join the cavernous sinus.

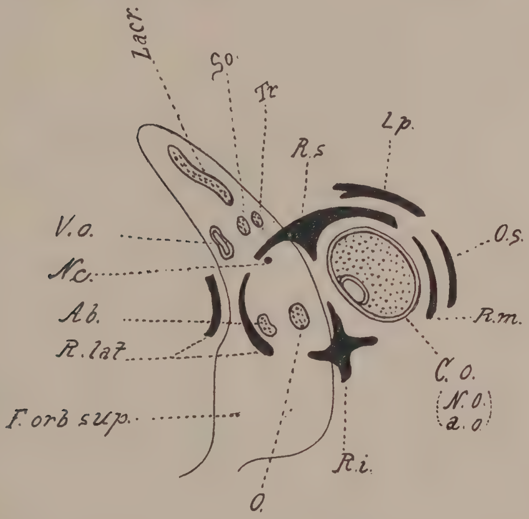

FIG. 2.—Schematic drawing of the structures at the apex of the orbit. (Merkel, *Topographische Anatomie*, Braunschweig, Vieweg, 1885.)

F.orb.sup., sphenoidal fissure; *R.lat.*, external rectus; *Ab.*, VI. nerve; *Nc.*, naso-ciliary nerve; *V.o.*, ophthalmic vein; *Lacr.*, lacrymal nerve; *S.o.*, supra-orbital nerve; *Tr.*, IV. nerve; *R.s.*, superior rectus; *L.p.*, levator palp.; *O.s.*, superior oblique; *Rect. med.*, internal rectus; *C.o.*, optic canal; *N.o.*, optic nerve; *A.o.*, ophthalmic artery.

OPTIC CANAL.—At the apex of the orbit the optic nerve passes through the optic foramen 1 *cm* long. At this point it enters into important relation with the sphenoidal sinus. A sphenoidal cell has been described in the lesser wing of the sphenoid. Onodi has drawn attention to an even more important relationship, in showing that frequently the posterior ethmoidal cells are also contiguous to the optic canal. This is particularly important because the intervening wall is usually very thin, much thinner than the sphenoidal wall, and so less likely to resist the extension of infection. One enlarged posterior ethmoidal cell may be in relation to both optic nerves.

The posterior central vein runs in the optic nerve backward and empties into the cavernous sinus. The ophthalmic artery lies within layers of the dura in the optic canal, so that no pressure can be exerted upon the optic nerve.

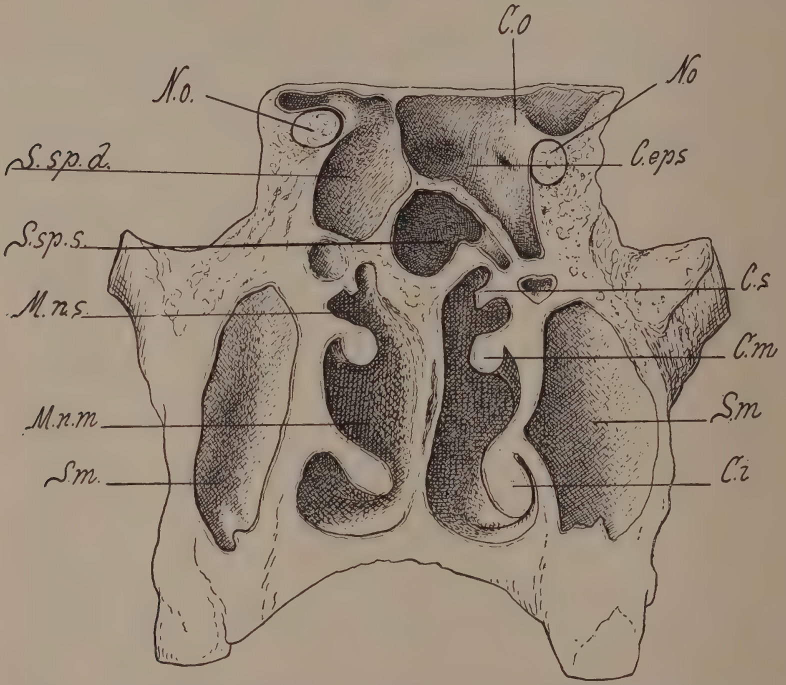

Fig. 3.—Transverse section of nasal fossæ showing relation of optic nerve to sphenoidal sinus and to posterior ethmoidal cells. (Onodi, *Der Sehnerv und die Nebenhöhlen der Nase.* A. Hölder, Wien u. Leipzig, 1907.)

C.o., optic canal; *N.o.*, optic nerve; *S.sp.d.*, right sphenoidal sinus; *S.sp.s.*, left sphenoidal sinus; *M.n.m.*, middle meatus; *S.m.*, maxillary sinus; *C.e.p.s.*, left post-ethmoidal cells; *C.s.*, superior turbinal; *C.i.*, inferior turbinal.

The close fit of the optic nerve in the canal causes it to be especially liable to a number of lesions: fracture through the canal wall leading to rupture of the optic nerve, periostitis, and narrowing of the optic foramen in tower skull. The canal is continued posteriorly by a fibrous ring composed of dura, which bridges over the upper part of the optic nerve. Below the optic nerve, at that point, is the internal carotid artery. The ophthalmic artery is given off here and passes directly forward to the optic canal. The close relationship of the ophthalmic artery to the lower surface of the optic nerve causes any change in its contour (atheroma) to press the optic nerve against the above-mentioned fold of the dura. The artery has been known to divide the nerve into two halves, or the pressure atrophy may be excentric. Wilbrand and Sänger believe these conditions deserve more consideration. An atheromatous in-

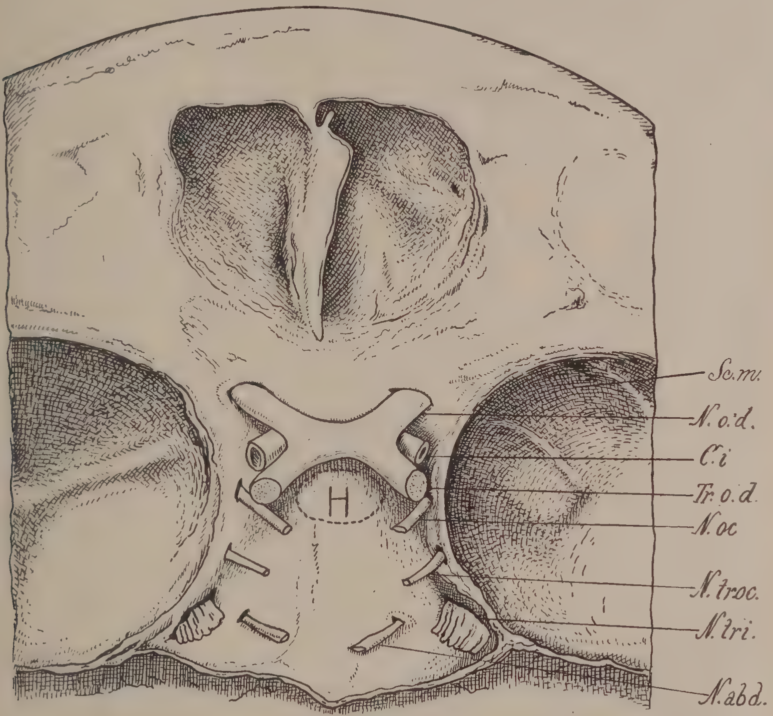

FIG. 4.—Base of skull showing entrance of nerves into dura. (Onodi, *Der Schnerv und die Nebenhöhlen der Nase.* A. Hölder, Wien u. Leipzig, 1907.)

Sc.m., middle cranial fossa; *N.od.*, optic nerve; *C.i.*, internal carotid; *Tr.o.d.*, optic tract; *H.*, hypophysis; *N.oc.*, oculomotor nerve; *N. troc.*, trochlear nerve; *N.tri.*, trigeminal nerve; *N.abd.*, abducent nerve.

ternal carotid may press the optic nerves upward and forward against the fibrous optic canal.

After leaving the optic canal, the optic nerve is situated within the cranial cavity for a distance of 1 *cm* and rests upon the dura and the convexity of the carotid artery. It is covered by the frontal lobe and the internal root of the olfactory nerve, from which it is separated by the

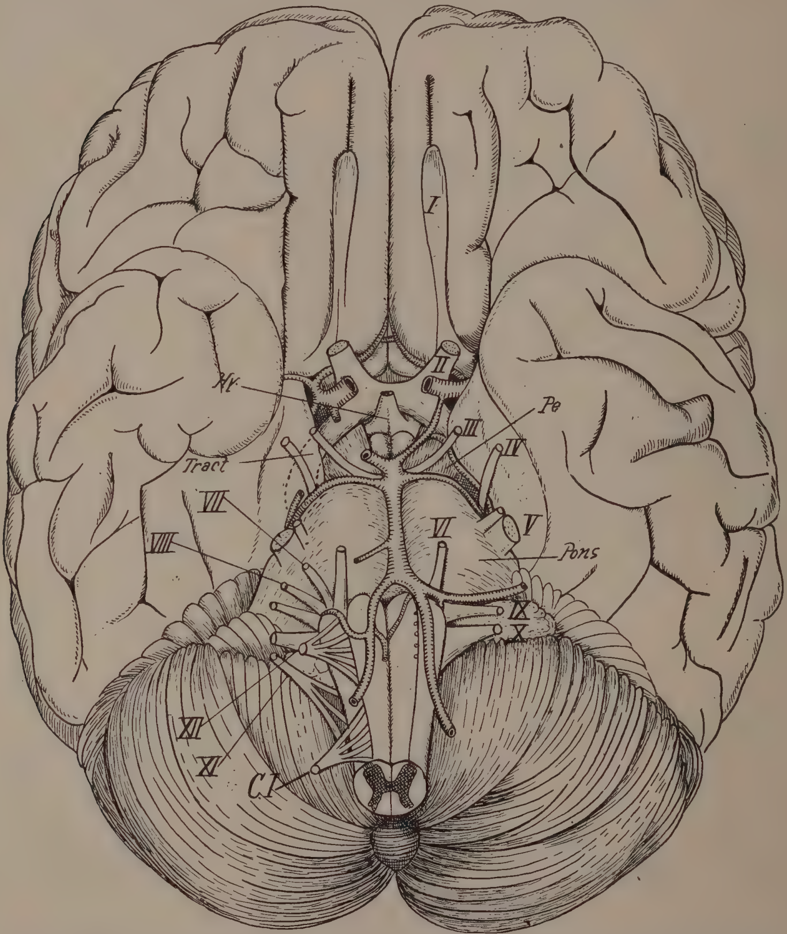

Fig. 5.—Base of brain showing nerves and arteries. (*Topographische Anatomie,* Bardeleben, Haeckel, Fischer, Jena, 1901.)
Hy., hypophysis; *Tract*, optic tract; *Pe*, peduncle..

anterior cerebral artery. The two optic nerves then converge to form the chiasm.

Optic Chiasm.—A study of the relation of the chiasm to the sphenoid body shows that the chiasm is situated back over the hypophyseal fossa, and not in front of it, and barely touches the tuberculum sellæ with its anterior margin. It does not touch the sphenoid below, and is separated from it by the cisterna chiasmatis. Pressure on the chiasm from below by a pituitary tumor causes resistance by the anterior communicating artery which is able to cut through the nerves. Enlargement of the pituitary body forward can attain 0.5 *cm* before the chiasm is pressed upon. Above the tubercle there is a furrow connecting the two optic foramina, which is called the sulcus chiasmatis, but which never contains the optic chiasm. At the lateral angles are the internal carotid arteries. At its posterior angle is the ventricular recess which extends forward over the upper-posterior half of the chiasm. Back and below its lower surface is the infundibulum.

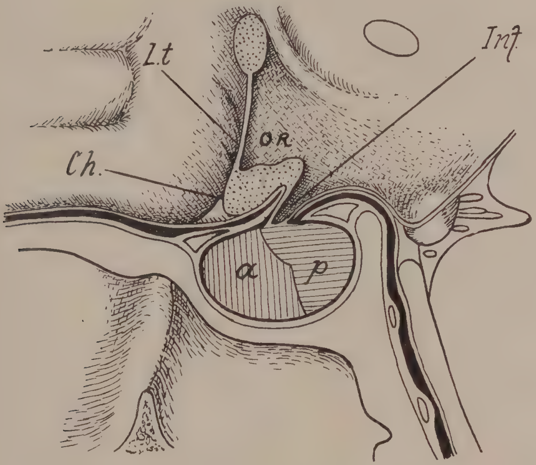

Fig. 6.—Hypophysis cerebri. (Testut.)
A., anterior lobe; *P.*, posterior lobe; *Inf.*, infundibulum; *Ch.*, chiasm; *L.t.*, lamina terminalis; *O.r.*, optic recess.

The posterior wall of the chiasm divides the aditus ad infundibulum into two: a lower part which extends below the chiasm to the infundibulum; and an upper part which lies above the chiasm. In man the anterior extremity of the recess extends to the middle of the chiasm. This very important recess can give symptoms under pathological conditions. Michel particularly drew attention to the importance of this recess. This cavity is in open communication with the third ventricle which, when distended, projects cyst-like over the anterior angle of the chiasm. Pressure from increased fluid would press upon the bilateral papillomacular bundles in the chiasm, causing a bitemporal paracentral scotoma, reaching to the point of fixation. Obscuration of vision in brain tumor may be due to sudden increase of the ventricular contents.

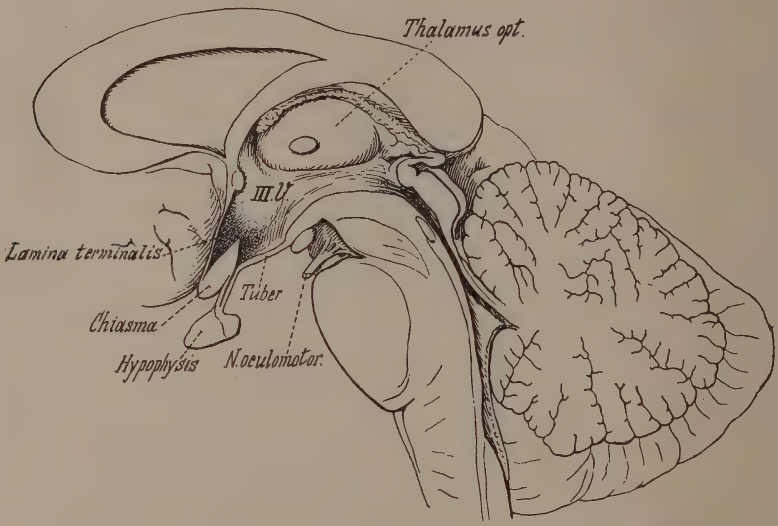

Fig. 7.—Sagittal section passing through chiasm and III. ventricle.

The chiasm is bathed in cerebro-spinal fluid; it separates the anterior confluent from the central confluent. The pia mater of the chiasm and its surrounding region is a site predilection for inflammation, especially tuberculous meningitis, cerebro-spinal meningitis and luetic meningitis.

Optic Tracts.—Posterior to the chiasm the optic fibres form the optic tracts which diverge from the chiasm outward and backward to surround the peduncles like spiral flat bands, and finally arrive at the optic ganglia. Anteriorly, for 15 *mm*, they lie free, posteriorly beginning at the anterior surface of the peduncles they are covered by the temporosphenoidal lobe for about 2 *cm*. The exposed part, together with the chiasm, is the part most affected in the various forms of meningitis. At the ventral surface of the thalamus each tract divides into two roots, the outer, or lateral root, passes to the external geniculate body and to the pulvinar, and the mesial root passes into the lower sur-

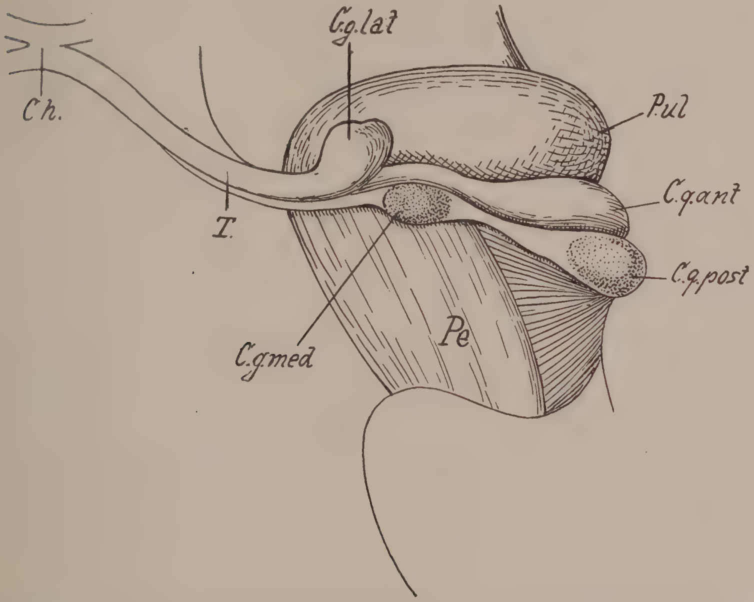

Fig. 8.—Primary optic centers: roots of the optic tracts: (Poirier, Charpy & Cuneo.)

Ch., chiasm; *T*, tract; *C.g.l.*, external geniculate body; *C.g.m*, internal geniculate body; *Pul*, pulvinar; *C.q.a.*, anterior quadrigeminal body; *C.q.p.*, posterior quadrigeminal body; *Pe*, crus cerebri.

face of the thalamus. Henschen has shown that the optic fibres throughout the optic paths assume and maintain a definite relation which is of the greatest importance for diagnosis. The termination of these two roots at the primary terminal stations of the optic fibres, constitute the primary optic centers.

Primary Optic Centers.—These number three:

1. The external geniculate body.
2. The pulvinar of the optic thalamus.
3. The anterior corpus quadrigeminum.

The primary visual centers have different functions. The external geniculate body is only a neuronal relay station in the passage of the visual fibres destined for the cortex. The anterior quadrigeminal body is a reflex center. It directs its neurons to the centers governing the movement of the iris and the other sensory and motor systems which are in connection with vision. The pulvinar sends possibly a few fibres to the cortex, but its principal function consists in connecting the visual apparatus with the other sensory and motor systems, particularly those which govern the expression of the face. Schematically one may say that the geniculate body is related to cortical and conscious vision; the quadrigeminal body to the reflex movements of visual origin; the pulvinar to automatic movements of visual origin, the latter two only giving unconscious perception. An animal deprived of its occipital lobe follows the light, evades obstacles, has normal pupillary reflexes, but does not recognize anything. In the lower animals these two centers are very much more developed than the first. In the more intelligent animals their size diminishes while the area of the cortex and of the external geniculate body increases.

The first, the external geniculate body, as has just been stated, is the chief primary optic center, the true between-brain of the optic path. It receives the greatest number (80 per cent.) of the fibres from the tract and sends visual stimuli through the fibres of the optic radiation to the visual sphere. According to Henschen, the individual optic bundles retain a definite position even in this ganglion and it can be said that the retina is projected on this ganglion. The dorsal segment must, therefore, correspond to the dorsal retinal quadrants of both eyes. Destruction produces constant and permanent quadrant hemianopsia in the lower halves of both fields.

Central Visual Path.—The central visual path or optic radiation arises in the external geniculate body, in the pulvinar and partly in the posterior portion of the anterior quadrigeminal body. The termination of the peripheric fibres and the origin of the central visual path are close together, so that impulses can be transmitted from one to the other. From the primary optic centers, principally the external geniculate body, the visual fibres pass through Gratiolet's optic radiation to the occipital cortex.

It was formerly believed that the mesial-most fibres of those passing to the occipital cortex, were the visual fibres, but to-day we know that the lateral fibres are the important ones for the optic radiation. This is the region which was called the fasciculus longitudinalis inferior, while the mesial part was regarded as the optic radiation. Both strata are now known as stratum sagittale occipitale, laterale et mediale. In the latter there are fibres of the occipital lobe (projection fibres) which pass on to the deeper parts; the fibres which belong to the optic radiation are situated in the former, the lateral layer, and are a bundle 1 *cm* broad. The dorsal fibres represent the dorsal part of the geniculate ganglion. The callosal radiation is internal to both in the wall of the ventricle. A lesion in this part causes a bilateral hemianopsia in the ventral quadrant of the field, and a lesion of the ventral fibres, hemianopsia of the dorsal quadrant. The macular fibres are isolated and each macula is connected with both cortical visual centers, though the position of the macular bundle is indefinite. Lenz believes that a typical macular preservation in complete interruption of conduction is only observed if the lesion is situated centrally to the internal capsule.

VISUAL CENTER—ANATOMIC VISUAL FIELD.—In the occipital lobe there is an anatomically sharply defined area (area striata) which from its position in the calcarine region undoubtedly is closely related to the central visual functions. This field possesses a very sharp boundary line. The optic fibres end in the cortex of the calcarine fissure which is known as the cuneus. The retina is here again pro-

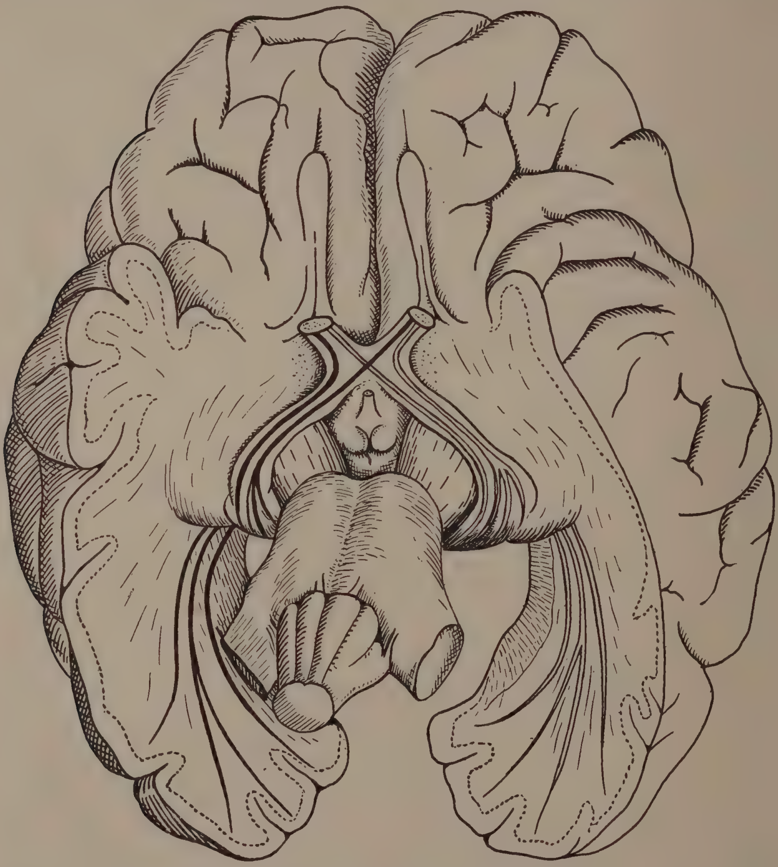

FIG. 9.—Visual paths. (Villiger, *Gehirn und Rückenmark*, III. ed., Leipzig, Engelmann, 1912.)

jected, and this region may be regarded as a definite negative of the retina (retina corticalis). The upper lip is the upper retinal half, the lower the lower half, the floor the horizontal line in which is situated the macula. Henschen places the macula in the depth of the fissure and in its anterior part which is unusually well supplied with blood vessels. Lenz places it in the posterior part. Each point of the macula has a bilateral representation.

Bilateral hemianopsia is not very rare; it occurs after an extensive bilateral lesion in the occipital lobe in the distribution of the arteriæ posteriores cerebri. Macular preservation is present at the onset of hemianopsia, the limits for color vision are parallel with those for white and vision is unaffected in the preserved parts (preëxisting double supply). Color perception is a higher function of the same elements (Lenz), hence one which is the first and the most easily affected. In bilateral hemianopsia the degree and extent of visual involvement vary from bilateral color hemianopsia to complete blindness in 25 per cent.

The cortical visual center transforms the physical image received on the retinal screen to a psychic one. The two cortical centers have but slight functional associations in animals (fishes) with only panoramic vision and the fields of the two eyes are independent, no point in space being seen simultaneously by the two eyes. There is therefore no binocular vision, and at the chiasm there is a total decussation of the optic nerves. In man vision is binocular, though in a part of each field objects can only be seen by one eye. Binocular vision permits us to receive and to fuse the two images that we obtain from the same object. Owing to the distance which the two eyes are separated, the object is seen from two angles. This gives us an impression of relief or that of the third dimension. It is this impression which enables us to judge of distance, particularly when the impression is aided by our knowledge of the degree of convergence and accommodation exercised.

The cortical centers are in relation with the centers of visual memory, also with other centers of special sense, the sensory paths and the motor paths. Their function in governing the ocular movements is evident, as the experiments of Mott and Shäfer have shown. The left cortical visual sphere which is in connection with the two left halves of the retina sees the right half of space. It commands the muscles which turn both eyes to the right. As Grasset[1] says, each hemisphere sees and looks at the opposite side. Thus a lesion of the visual sphere may produce half vision or half blindness of the opposite side, and a conjugate deviation of the head and of the eyes to the side affected (predominence of the antagonists). The cortical center of the elevator of the upper eye lid in the angular gyrus is also closely connected. There are other important associations with the cerebellum in aid of our sense of orientation in which the eyes and the semicircular canals act as principal forces.

Visual Agnosia,[2] *Mind blindness*, is the result of a lesion of the psycho-sensorial paths. The receiving and transmitting organs are normal, the higher or conscious sensations are affected. Visual agnosia constitutes psychical blindness. There are several varieties: (1) Complete psychical blindness. The patient is no longer able to recognize anything about him. He sees objects but their image does not awaken any impression. (2) Optic aphasia of Freund. The patients recognize objects, their use and their characters, but have lost the ability to name them. (3) Verbal blindness or alexia. Writing or printed text does not signify anything to the patient any more than the text of a foreign language. The text will be recognized as such and the book will be turned if it is handed to the patient upside-down, though this is sometimes not noticed. The writing is like that of a person

[1] Grasset, quoted from de Lapersonne et Cantonnet, Neurologie oculaire. Paris, Masson et Cie., 1910.

[2] de Lapersonne et Cantonnet, l. c.

who keeps his eyes closed and will not be able to read what he has written. This may affect letters, which are no longer recognized (letter blindness), or the letters are recognized but the faculty to assemble them in syllables or in words is lost (syllable or verbal blindness). The patient will sometimes succeed in reading if he follows each letter with his finger. If he has to copy a word he will do it as if it were a drawing. Sometimes the reading of words is correct while the reading of music becomes impossible (musical blindness). Verbal blindness is often accompanied by verbal deafness (sensory aphasia of Wernicke). Dyslexia (Bruns) is an analogous trouble to verbal blindness, except that it occurs a certain length of time after reading and then disappears with rest; in other words, it comes and goes.

If the lesion of the left cortical visual sphere extends to the visual memory center, verbal blindness results which is always associated with right homonymous hemianopsia. The opposite condition does not hold. This association is explained by the fact that the lesion involves the visual memory center on the convex surface of the hemisphere and the subcortical layers in which are found the optic radiations. If the lesion which produces verbal blindness is truly cortical, there is also verbal deafness, sensorial aphasia of Wernicke. The deviation of the eyes to the side opposite of the hemianopsia (paralysis of the motor forces which turn the eyes to the right) may be present because the visual cortical center is also the posterior center for the movements of the eyes. This is, however, unusual, as the anterior center is the more active.

Course of the Optic Fibres.—In the peripheric visual path we can distinguish the following groups of fibres: a crossed, an uncrossed and a macular bundle. The crossing of the fibres takes place in the chiasm where the optic nerves undergo a partial decussation. A small part of the fibres of each optic nerve pass through the chiasm to the tract of the same side and a larger bundle of fibres (75 per cent.

v. Monakow) crosses to the tract of the opposite side. This results in the retina being connected with both cerebral

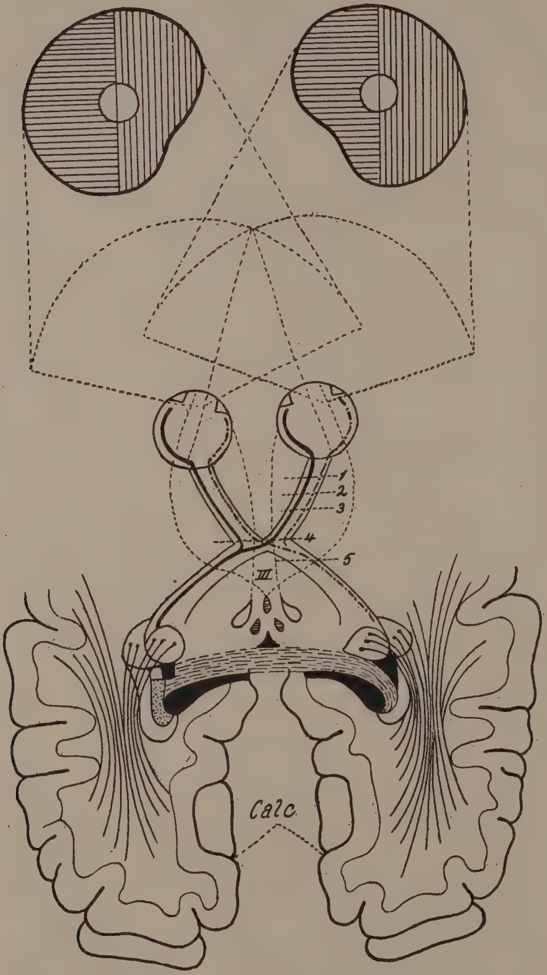

FIG. 10.

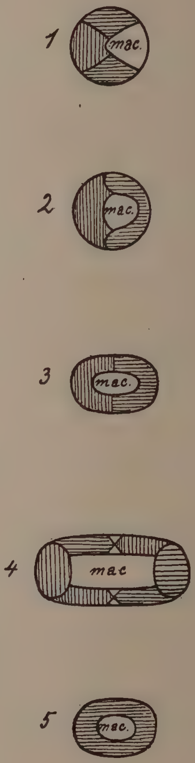

FIG. 11.

FIG. 10.—Visual paths, showing position of fibres. (Axenfeld, Lehrbuch d. Augenheilk., Fischer, Jena, 1914.)

FIG. 11.—Right optic nerve.

1, Posterior to eyeball; 2, half-way between eyeball and chiasm; 3, just anterior to chiasm; 4, chiasm; 5, tract.

hemispheres. Each tract and visual center receive fibres from the eye of the same side and of the opposite side, though each tract contains only fibres from the homonymous retinal halves of both eyes.

In the chiasm the fibres from the nasal part of both retinæ cross each other, while the temporal ones continue uncrossed. The crossing fibres describe an S-shaped figure in the chiasm. The chiasm also contains fibres which are not visual fibres. At its dorsocaudal part there is Gudden's commissure, a connection between the two internal geniculate bodies. Directly above this there is Meynert's commissure, whose relation to the lenticular nucleus and corpus subthalamicum is definite. There are also a few fibres of the commissura hypothalamica. In addition to these there are transverse fibres whose relation to the optic nerves is not known (commissura ansata anterior and posterior).

The macular fibres arise from the macula lutea and form a closed wedge-shaped bundle in the ventro-lateral part of the optic nerve with its broad end to the periphery. As it proceeds back it becomes semilunar and reaches the center of the optic nerve at about 15 to 20 *mm* posterior to the eyeball. This bundle passes directly backward in the central part of the optic nerve as a vertical then horizontal oval, retains its central position in the chiasm and in the tract. The macular fibres undergo a partial decussation in the chiasm, and both tracts and primary optic centers contain a crossed and an uncrossed macular portion.

The uncrossed direct bundle arises from the temporal retinal halves of both eyes, and everywhere occupies the external (temporal) side of the optic paths. Directly behind the papilla the bundle is divided in a ventral and a dorsal half by the interposition of the macular fibres; the fibres then unite more centrally.

The crossed bundle originates from the mesial (nasal) retinal halves of both eyes, and occupies the internal, that is, nasal periphery of the optic nerve, chiasm and optic tract.

In addition to the actual visual fibres, the optic nerves possess centrifugal fibres which serve for the optic reflex. The ones which serve the pupillary reflex paths are alone of importance. The arrangement of the pupillary fibres in the optic nerve is not understood. They are probably mixed with the other fibres and partially decussate in the chiasm. From the tract the pupillary fibres pass with the mesial optic root to the anterior quadrigeminal body. From there the reflex is transmitted to the oculomotor nucleus.

It is generally accepted that the various optic bundles and fibres occupy a constant position in their course through the peripheric visual path. Clinical observations have shown that the fibres of the dorsal retinal quadrant are always situated dorsally, and the ventral quadrants correspond to the ventral position in the visual path. Observation of hypophyseal tumors have shown that the fibres of the dorsal macular half are situated above, while the ventral half is situated below in the macular bundle.

Topographic Diagnosis.—If we now endeavor to trace the symptoms which lesions in the various parts of the optic paths produce, we find:

1. Optic nerve. Homolateral amaurosis. Homolateral loss of direct, contralateral of consensual pupillary reaction ("Reflex Taubheit").
2. Chiasm. Median part. Bitemporal hemianopsia (acromegaly, dystrophia adiposo-genitalis).
3. Only in bilateral affections. Bilateral nasal hemianopsia (arteriosclerosis, lues, trauma).
4. Tract. Hemianopsia of definite character including macula, hemianopic iridoplegia (hemiplegia, same-sided ptosis (Wernicke's syndrome).
5. If only the ventral or dorsal part of the geniculate body is affected then quadrant hemianopsia.
6. Hemianopic iridoplegia.
7. Bilateral homonymous hemianopsia (in so-called carrefour sénsitive with hemihypæsthesia and hemiplegia; capsule lesion).

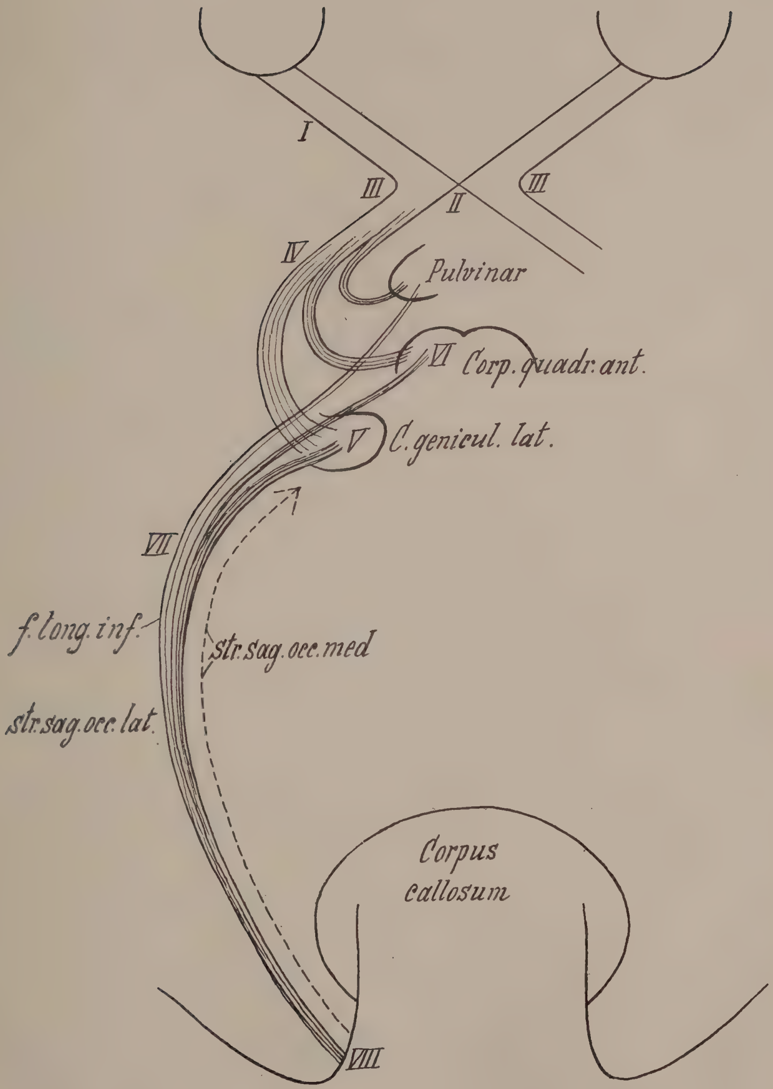

FIG. 12.—Topographical diagnosis of visual path lesions.

8. Right like (7). Bilateral cortical blindness. Left, soul-blindness.

It is evident that these disturbances occur only after severe lesions of the cortex. It is possible that only certain fibres are destroyed which connect the calcarina with other parts of the brain. Hence in left-sided foci there may be disturbances of speech (optic aphasia, alexia), or disturbances of color perception (hemiachromatopsia), or of form.

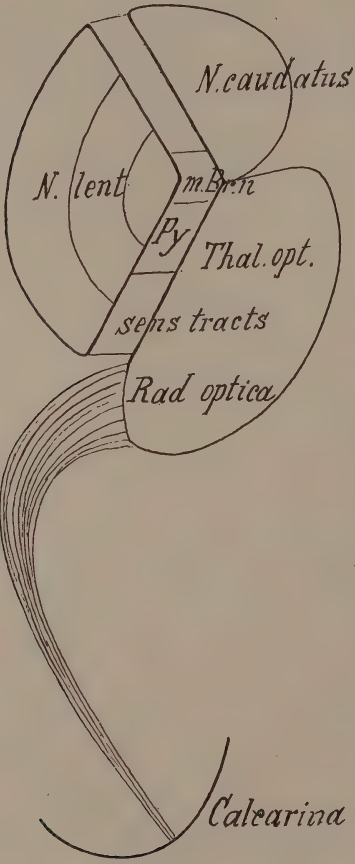

FIG. 13.—Topography of internal capsule. *M.b.n.*, Cerebral motor nerves.

OCULAR NERVES

The nerves which control ocular movements were originally mixed nerves. In the course of evolution they lost their sensory fibres, which have gone to form the sensory part of the trigeminal nerve. In addition to the oculomotor (III), trochlear (IV), and abducent (VI), we must speak of the facial on account of its rôle in the movements of the eye lids, and of the sympathetic on account of its function connected with the iris. The trigeminal and vestibular nerves are also of importance as accessory nerves. The roots of the III, IV, and VI nerves are situated beneath the floor of the fourth ventricle and of the Sylvian aqueduct.

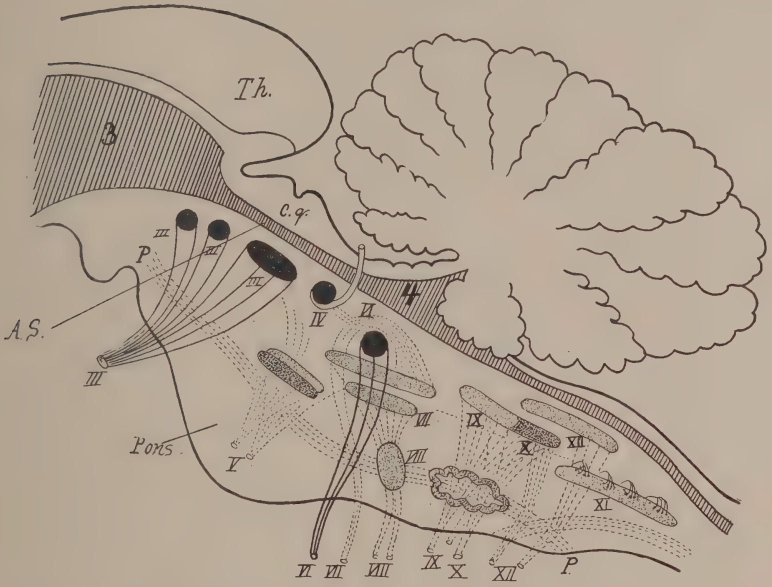

FIG. 14.—Diagrammatic sagittal section to show nuclei of origin of the nerves of the ocular muscles. (Knoblauch, *Allgemeine Chirurgie d. Gehirnkrankheiten v. Krause.* Enke, Stuttgart, 1914.)

Th., thalamus; *Q*, corpora quadrigemina; *PP.*, pyramidal tracts; *P.*, pons.

Roots.—The *oculomotor nucleus* is composed of (1) a single nuclear column situated in the median line whose cells increase as they pass from the oral to the caudal segment (nucleus medialis); (2) of a large-cell lateral or main nucleus which can be divided into a medio-ventral and a dorso-lateral division; and finally, (3) there is a nucleus situated between the above two, rather nearer to the anterior extremity of the nucleus, surrounded by a pale ground-substance and is called the Edinger-Westphal nucleus. In addition to these three main nuclei, there are a number of isolated cells which are situated on the lateral side of the lateral nucleus. All other cells situated about the aquædductus Sylvii have nothing to do with the oculomotor nerve. The cell column of the oculomotor nerve measures about 1 *cm* in length and is continuous in a caudal direction after a short interruption with the round nucleus of the trochlear nerve.

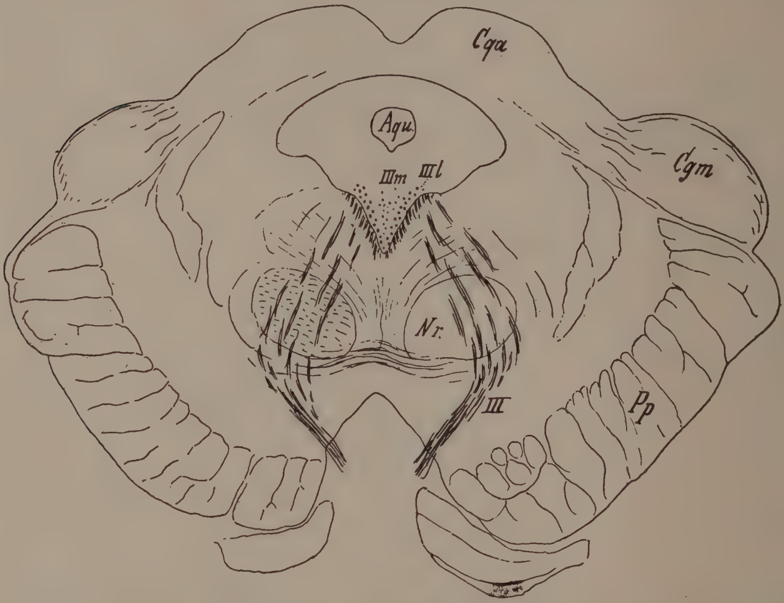

FIG. 15.—Oculomotor nucleus.

C.q.a., anterior quadrigeminal body; *C.g.m.*, internal geniculate body; *III.m.*, internal oculomotor nerve; *III.l.*, external oculomotor nerve; *N.r.*, nucleus ruber; *P.p.*, pes pedunculi; *III.*, oculomotor nerve; *Aqu.*, aqueduct of Sylvius.

The *trochlear nerve root* is found where the posterior corpora quadrigemina join the anterior corpora. On the caudal side of this nucleus there are the cells of Westphal-Siemerling, which have no relation to the ocular muscles. The round nucleus of the *abducent nerve* is situated directly in front of the middle of the floor of the fourth ventricle, quite near the median line. This nucleus possesses an accessory part which is situated on the ventral side and laterally near the facial nerve (accessory abducent nucleus). Its significance is not understood.

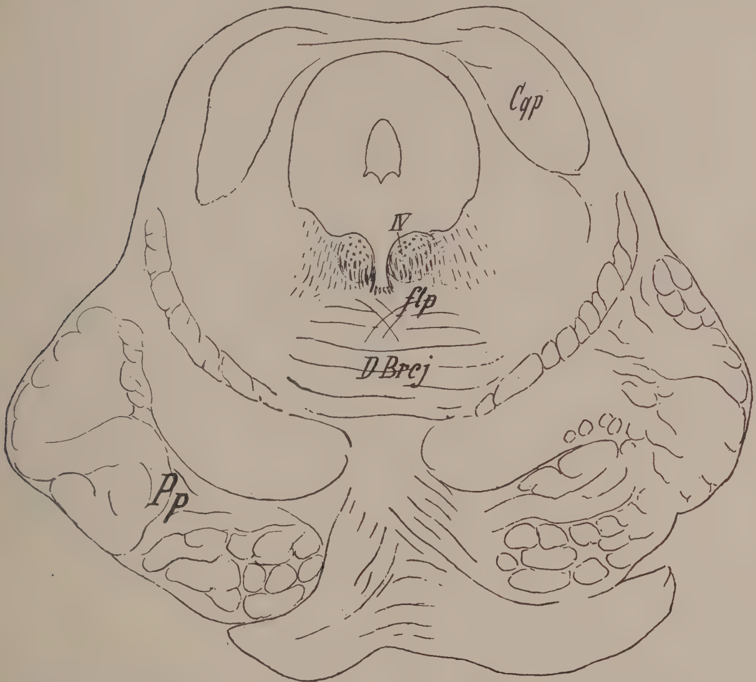

FIG. 16.—Trochlear nerve nucleus.
C.q.p, posterior quadrigeminal body; *IV*, trochlear nerve; *F.l.p.*, posterior longitudinal bundle; *Dbrci*, decussatio braccio conjunctivorum; *P.p.*, pes pedunculi.

The relation of the individual eye muscles to these nuclei is clear as far as the abducent and trochlear nerves are concerned: the former innervates the external rectus of the same side, while the superior oblique of the opposite side is innervated by the latter, as its fibres cross before they leave the brain.

The relation of the various parts of the *oculomotor nucleus* to the muscles is somewhat more complex. We know that the anterior and internal part of the nucleus governs the intrinsic muscles and that the majority of the fibers are homolateral or direct, while the other parts which cross to the internal and inferior recti and inferior oblique are crossed. As the intrinsic muscle nuclei are grouped in the middle line separated from the others and with a distinct blood supply, it seems probable to assume that a vascular lesion may affect either the extrinsic or the intrinsic muscles at the same time. Their close relationship ex-

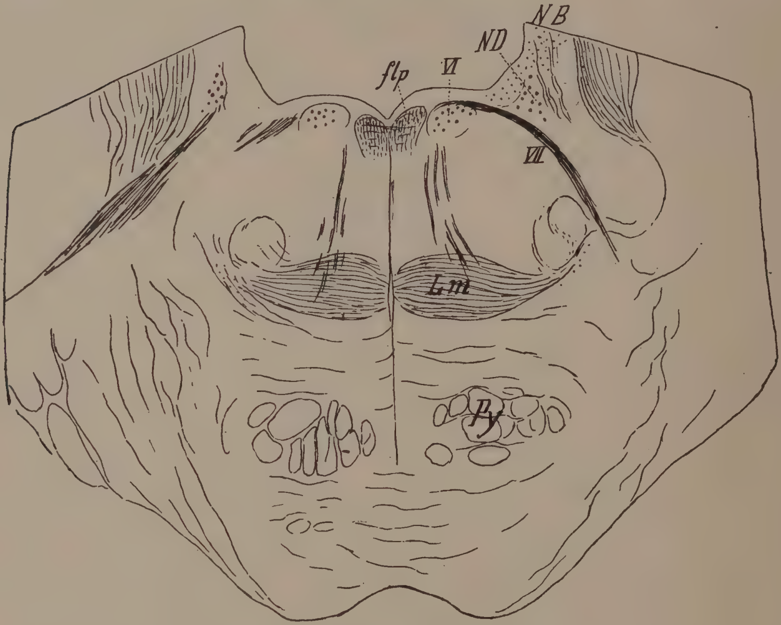

FIG. 17.—Pontine nuclear region.

F.l.p., posterior longitudinal bundle; *VI*, abducent nucleus; *N.D.*, Deiter's nucleus; *N.B.*, Bechterew nucleus; *VII*, facial nerve; *L.m.*, mesial fillet (lemniscus medialis); *Py.*, pyramidal tract.

plains their associated functions, their independent blood supply their isolated lesions. The grouping near the middle

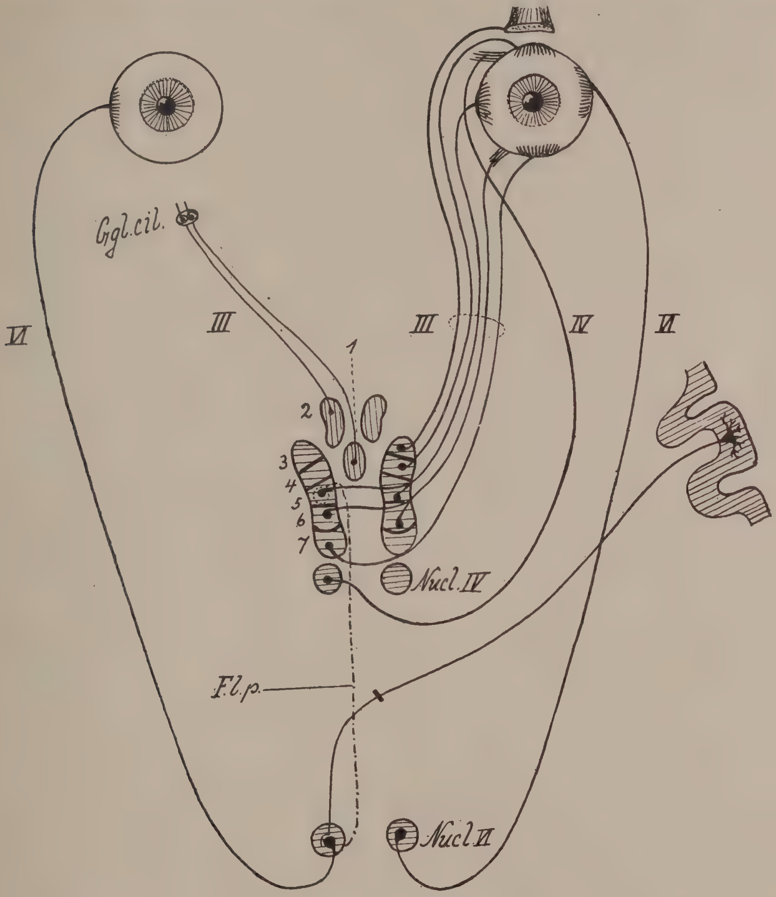

FIG. 18.—Innervation of the ocular muscles. (Bing, *Kompendium d. topischen Gehirn und Rückenmarksdiagnostik,* II. Auf., Berlin, Wien. Urban u. Schwarzenberg, 1911.)

G.c., ciliary ganglion through which pass the nerves to the internal eye muscles. 1, small cell median nuclear center for ciliary muscle; 2, small cell lateral nucleus center for sphincter pup.; 3, 7, large cell lateral nucleus; 3, levator palp. sup.; 4, rectus sup.; 5, rectus int.; 6, obliquus inf.; 7, rectus inf.; *III*, oculomotor nerve; *IV*, trochlear nerve; *VI*, abducent nerve; *F.l.p.*, posterior longitudinal bundle.

line of the nuclei of the intrinsic muscles explains their bilateral association (accommodation and pupillary contraction are always bilateral) and their association among themselves (accommodation is always accompanied with contraction of the pupil). Without doubt the lateral nucleus serves the external muscles and the part most orally and laterally supplies the levator palpebræ. Then comes the rectus superior, the rectus inferior and the obliquus inferior. The mesial nucleus is divided into two parts, as has already been described. The caudal division belongs to the internal rectus as far as it serves convergence, while the part situated in the lateral nucleus serves the internal rectus, for its function in associated movements. The

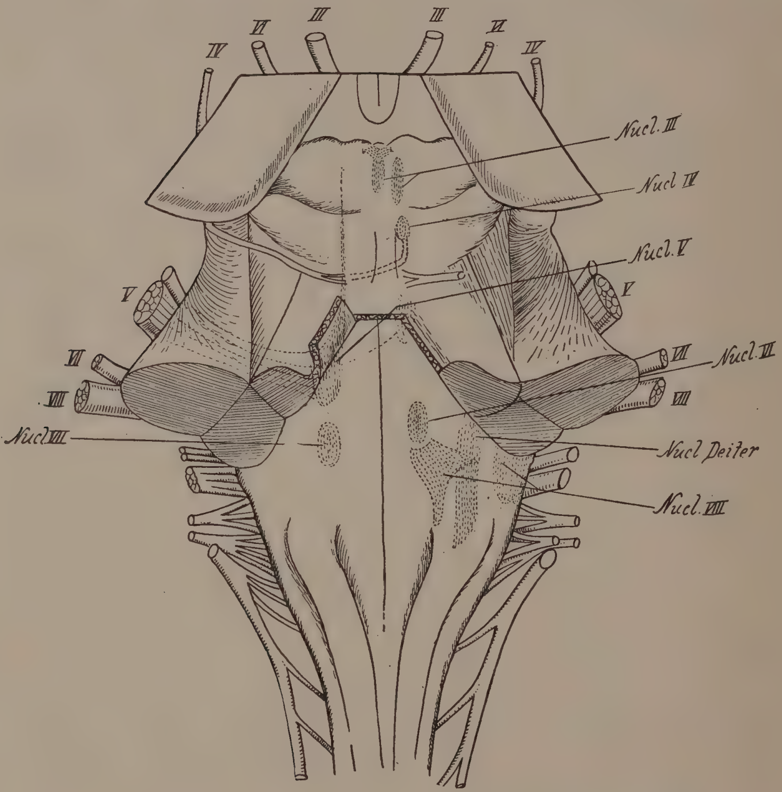

FIG. 19.—Dorsal view of the floor of the fourth ventricle, showing the location of the nuclei of the ocular nerves. (After Ziehen, *Topographische Anatomie*, Bardeleben, Haeckel, Fischer, Jena, 1901.)

anterior part of the mesial nucleus serves principally the pupil, which means primarily the light reaction of the pupil. Opinions differ on the importance of the Edinger-Westphal nucleus; we know only that it belongs to the oculomotor nerve.

The root fibres pass outward from this oculomotor nucleus, crossed and partly uncrossed. All fibres of the mesial nucleus are uncrossed. The fibres of the lateral nucleus are crossed and, in fact, partially crossed, especially the fibres from the lateral nucleus to the internal rectus are crossed. It has already been mentioned that the trochlear fibres decussate. The decussation takes place in the velum medullare anterius. The fibres of the abducent nerve leave the nucleus on its mesial side. They do not pass directly out to the ventral surface, but are directed somewhat caudally in order to reach the base of the pons near the middle line. While the two last-mentioned nerves form enclosed bundles, the oculomotor nerve splits up into a number of small bundles which are separated and do not join together until just before their exit at the peduncle or just beyond this point sometimes at a distance of ½ *cm.*

The ocular muscle nuclei have important relation to the principal conducting paths and to the nuclei of the mid-brain and of the medulla oblongata. The fasciculus longitudinalis posterior is closely related to the oculomotor nucleus and is indented by the trochlear nucleus, while the abducent nucleus is situated on its lateral surface. In the mid-brain the nucleus ruber comes into relation on its ventral surface with the oculomotor fibres which pass through it. Furthermore should be mentioned the tracts of the tegmentum (sensory paths) and those of the pes pedunculi, *i.e.*, the motor paths in this as well as in the pons.

The SYNDROMES which are produced by a combined lesion of the ocular muscles and of the above-mentioned nuclei and tracts are the following:

1. Mid-brain.

(*a*) Weber's syndrome. Site of lesion: Pes pedunculi.

Homolateral oculomotor paralysis, controlateral hemiplegia. The oculomotor paralysis is generally complete, except when the fascicles unite somewhat late, as mentioned above, when it is incomplete.

(*b*) Syndrome of Benedikt. Homolateral fascicular oculomotor paralysis (partial), contralateral athetosis, chorea or tremor (of the character of paralysis agitans) lesion in the region of the nucleus ruber.

(*c*) Syndrome of Nothnagel. Nuclear (bilateral) partial oculomotor paralysis and ataxia. Lesion: region of the aqueductus and the roof of the corpora quadrigemina.

These syndromes refer only to these regions when development of the symptoms occurs simultaneously or directly one after the other. It is important for the last of the three mentioned syndromes to remember that the ocular paralysis is primary and that the ataxia occurs secondarily, as in the reverse case the lesion may be situated in the cerebellum and then exert pressure upon the corpora quadrigemina (Bruns). The optic tract passes laterally around the cerebral peduncle. It occurs occasionally that it is affected together with the neighboring regions by trauma (penetrating wound), tumor or lues. The syndrome of Wernicke, which corresponds to this localization, is homolateral ptosis, contralateral hemiplegia and tract hemianopsia. The abducens may be affected in the pes pedunculi as well as in the tegmentum. In the former case there is homolateral abducens paralysis and contralateral hemiplegia (syndrome de Raymond). The lesion in the pons more frequently affects the facialis (abducens) of the same side and hemiplegia of the opposite side (syndrome de Millard-Gubler). The abducens is more rarely affected in a lesion of the tegmentum. In place of an abducens paralysis there is a homolateral paralysis of associated ocular movement (syndrome de Foville-Wernicke).

PERIPHERIC COURSE.—**The III nerve** fibres pass through the thickness of the peduncle in a curved path along the internal aspect of the posterior longitudinal bundle and of the nucleus ruber, and emerge by about 30 roots, one half directly coming out of the peduncle, the other half out of the interpeduncular space. The fibres re-unite to form the trunk of the nerve whose neurilemma is formed by a reflection of the pia. This nerve travels in the subarachnoid space, passing between the peduncle posteriorly and the

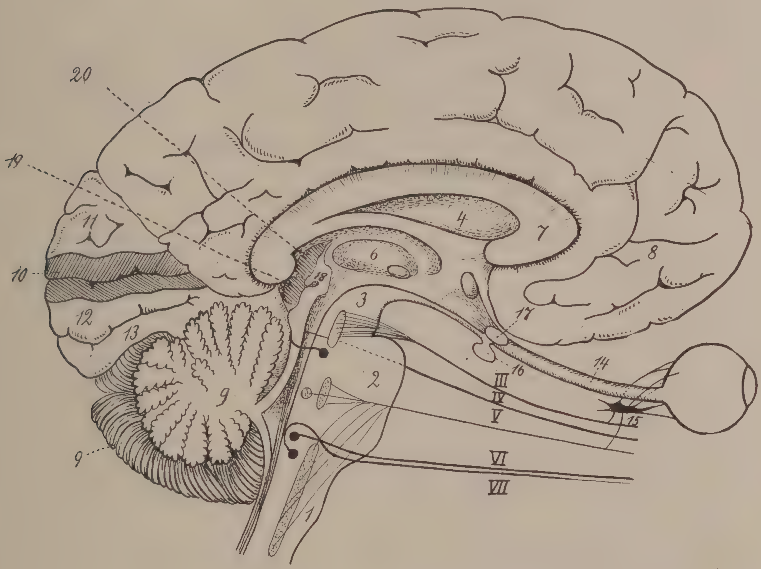

Fig. 20.—The relation of the ocular nerves to the brain. (From de Lapersonne et Cantonnet, *Manuel de Neurologie Oculaire*, Masson et Cie, Paris, 1910.)

1, medulla; 2, pons; 3, peduncle; 4, septum lucidum; 5, infundibulum of 3d ventricle; 6, optic thalamus; 7, corpus callosum; 8, frontal lobe; 9, cerebellum; 10, calcarine fissure (left); 11, cuneus; 12, lingual gyrus; 13, fusiform gyrus; 14, optic nerve (left); 15, ciliary ganglion with its connections; 16, pituitary body; 17, section of chiasm; 18, pineal gland; 19, external geniculate body; 20, pulvinar; *III*, *IV*, *V*, *VI*, *VII*, cranial nerves.

basilar apophysis in front. During this course the posterior cerebral artery passes above and the superior cerebellar artery below. It perforates the dura mater which by the junction of the two circumferences of the tent of the cerebellum forms the external wall of the cavernous sinus. It travels in this wall in company with the other nerves and enters the orbit through the internal part of the sphenoidal fissure. At the moment of its penetration it bifurcates, giving off a superior branch and an inferior branch, one above and one below the optic nerve, the upper

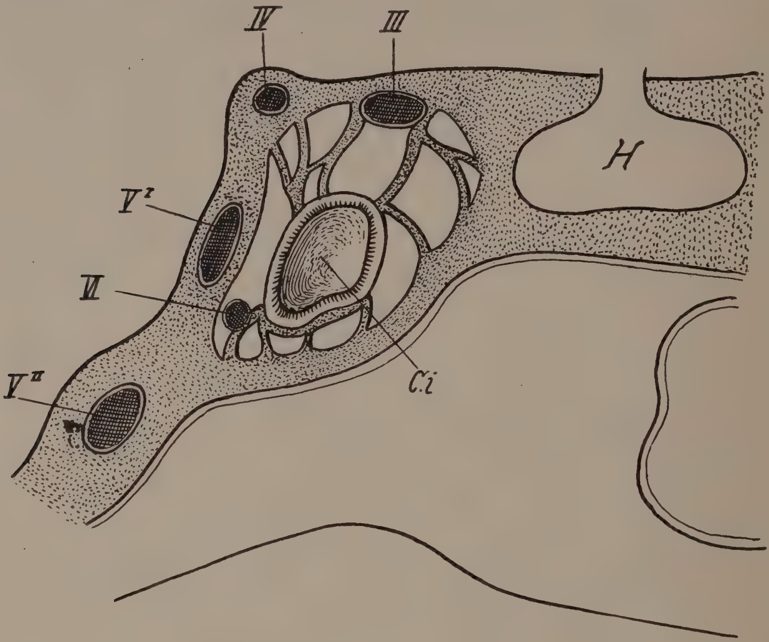

FIG. 21.—The nerves in the outer wall of the cavernous sinus. (Knoblauch, *Allgem. Chirurgie d. Gehirnkrankheiten*, Krause, Enke, Stuttgart, 1914.)
H, pituitary body; *C.i.* internal carotid; *III*, oculomotor; *IV*, trochlear; *VI*, abducent; V^I, ophthalmic nerve; V^{II}, superior maxillary nerve.

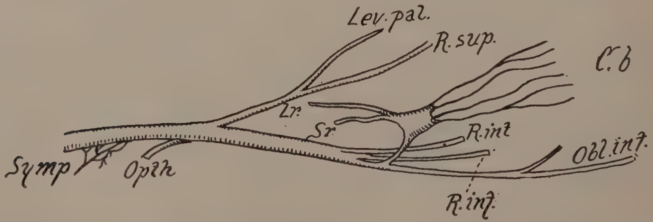

FIG. 22.—Diagram of the III. nerve with the ciliary ganglion. (G. D. Thane, *Quain's Anatomy*.)
L.r., long root; *S.r.*, sympathetic root; *C.b.*, ciliary branches.

one supplying the rectus superior and the elevator of the lid, the lower one being distributed to the internal rectus, inferior rectus and inferior oblique. The branch to this last muscle gives an anastomatic branch to the ciliary ganglion.

The IV nerve takes its origin by roots from its peduncular nucleus and differing from the other nerves which are directed ventrally, it passes to the dorsal aspect of the nerve trunk. They are obliquely inclined to the median line and decussate within the interior of the peduncle. The nerve emerges at the level of the valve of Vieussens, below the posterior quadrigeminal body. This dorsal exit and its decussation are two features by which this nerve differs from all the other cranial nerves. In order to proceed anteriorly, the nerve must pass around the lateral face of the peduncle to which it is adherent. During this passage it is above the superior margin of the pons and below the optic tract. It travels in the subarachnoid space, perforates the outer wall of the cavernous sinus below the III nerve, and enters the orbit in the supero-external portion of the sphenoid fissure. It travels in the upper part of the orbit and attains the superior margin of the large oblique muscle.

The VI nerve passes directly through the thickness of the pons external to the pyramidal tract. It emerges in the depression between the pons and the medulla, above the pyramids and internal to the facial nerve. The middle cerebellar artery passes either above or below it. This nerve leaving the nervous trunk somewhat below the III and IV pairs, perforates the dura mater somewhat lower down, not at the tentorium but below it just at the upper border of the petrous pyramid which it crosses as it passes through the middle cranial fossa. It is in direct contact with the osseous articulation to which it is closely applied by the superior petrosal sinus and the petrosphenoidal ligament, a fibrous ring stretching from the apex of the pyramid to the posterior clinoid apophysis. In cranial injuries where the petrous pyramid becomes ever so slightly loosened, the nerve may become injured (Panas). Also, osteitis of the apex of the pyramid may be accompanied with the paralysis of the VI nerve. The nerve does not penetrate the outer wall of the cavernous sinus the way the other nerves do, but comes to be placed within the cavity of the sinus, bathed in the venous blood and close to the external surface of the carotid artery, surrounded by the pericarotid sympathetic plexus. After passing through the largest part of the sphenoid fissure, the abducent nerve passes along the outer part of the orbit and enters the rectus externus muscle.

Fisher[1] says: "Exaggerated notions of the length of the cranial nerves between their superficial origins and the points at which they perforate the dura mater are apt to arise; this portion of the III nerve measures little more than one inch; the corresponding part of the small IV nerve takes a very circuitous route, but is not longer than two inches; of the VI nerve the length of this intradural part is about an inch and a quarter. It is thus seen that only for a comparatively short distance are these nerves exposed to implication in meningeal disease."

Other motor and sensory nerves which are related to the movement of the eyes are the following:

Facial Nerve.—The nucleus of the facial nerve is situated ventrally and somewhat caudally from the abducent nucleus. It sends its fibres in a dorso-mesial direction to the floor of the fourth ventricle and from where it at first passes orally and then in a curved direction laterally and ventrally. The abducent nucleus is situated within the knee produced by this course. The nuclear column of the facial can be distinctly divided into numerous groups of cells of which the dorsal one belongs to the ocular facial. The secretory fibres of the facial nerve are derived from the nervus intermedius Wrisbergii, which is supposed to spring from the large cells in the tegmentum (nucleus salivatorius).

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[1] Fisher, Ophthalmological Anatomy, London, 1904.

The association of the nucleus of the facial nerve to other nerves is of great importance. It is claimed by some that the ocular facial is derived from the oculomotor nucleus, while the lower facial is derived from the hypoglossal nucleus. It has a close relation with the V nerve. It emerges at the level of the lateral depression of the pons, external to the III nerve and internal to the auditory nerve. It enters the internal auditory meatus and as it passes through the petrous pyramid it presents a thickening, the geniculate ganglion. It finally passes forward through the parotid gland. One anastomosis of the facial

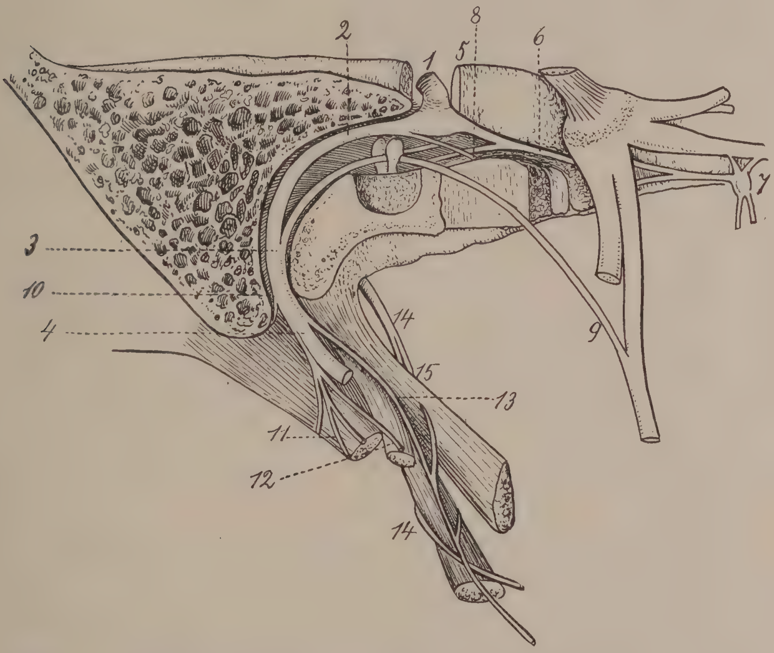

FIG. 23.—The facial nerve in its canal, with its connecting branches, etc. (From Sappey, after Hirschfeld and Leveillè, *Quain's Anatomy*, Vol. III, Pt. II.)

1, facial nerve in the first horizontal part of its course; 2, its second part turning backwards; 3, its vertical portion; 4, the nerve at its exit from the stylomastoid foramen; 5, geniculate ganglion; 6, large superficial petrosal nerve; 7, spheno-palatine ganglion; 8, small superfical petrosal nerve; 9, chorda tympani; 10, posterior auricular branch cut short; 11, branch to the digastric muscle; 12, branch to the stylohyoid muscle; 13, twig uniting with the glosso-pharyngeal nerve (14 and 15).

is of interest to us, and that is the great superficial petrosal nerve which unites the facial nerve with the sphenopalatine ganglion, a part of the superior maxillary nerve.

In determining the seat of a facial paralysis the involvement of the orbicularis muscle is of particular diagnostic importance. In cortical and sub-cortical affections of the facial nerve the lower branches of this nerve are alone affected. A nuclear or peripheric paralysis generally produces a complete facial paralysis. The electrical examination is of importance. The facial paralyses which are of nuclear origin are usually bilateral, as is explained by the proximity of the nuclei. In tabes where the nuclei of the cranial nerves are so frequently involved a facial paralysis is very unusual. On the other hand, a nuclear lesion of the facial nerve is more frequent in acute superior hemorrhagic polioencephalitis. In lesions of the pons the involvement of the facial nerve is of interest as the characteristic paralysis for a pontine lesion consists in an alternating paralysis with involvement of the facial nerve on the same side as the lesion and a paralysis of the opposite extremity. This is the condition when the focus lies above the pyramidal crossing but below the facial crossing. If the focus is

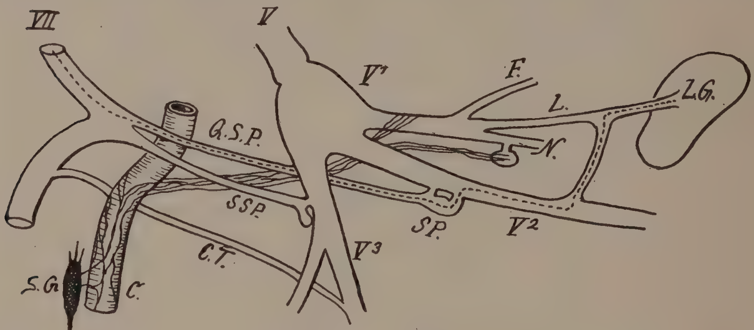

FIG. 24.—Connections of the facial and trigeminal nerves. (de Larpersonne et Cantonnet, *Neurologie oculaire*, Paris, Masson & Cie, 1910.) Dotted line shows facial innervation of lacrymal gland.

VII, facial nerve; *G.P.S.*, great superficial petrosal; *P.P.S.*, superficial petrosal; *C.T.*, chorda tympani; *C*, internal carotid artery; *V.*, trigeminal nerve and Gasserian ganglion; *V*¹, ophthalmic nerve; *F.*, frontal; *L.*, lacrymal; *N.*, nasal and ophthalmic ganglion; *G.L.*, lacrymal gland.

above the facial crossing then of course the paralysis of the facial and of the extremities are on the same and opposite sides (Gubler paresis).

A paralysis of the peripheric facial nerve is simple because all the branches are involved. By referring to the accompanying Erb's diagram the following main types can be distinguished:[1] (1) Paralysis of the facial muscles with salivary secretion, taste, hearing and soft palate intact; (2) paralysis of the facial muscles, disturbance of taste and of salivary

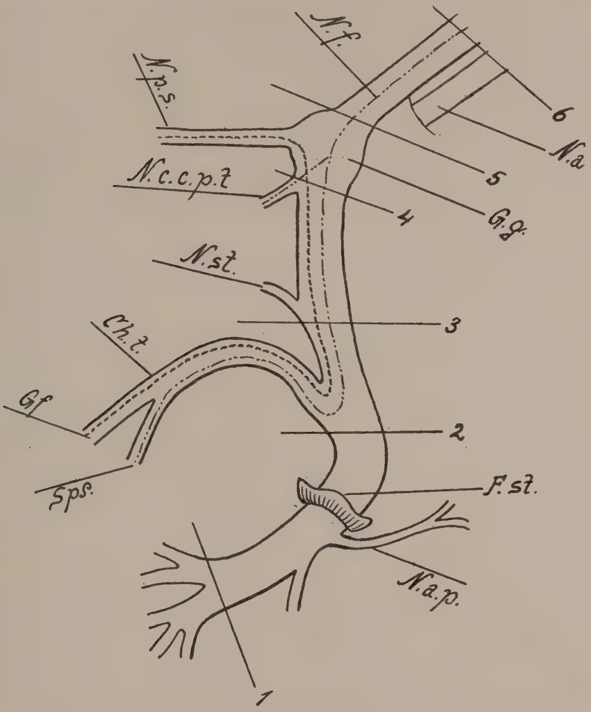

FIG. 25.—Schematic drawing of the facial nerve from the base of the skull to the pes anserinus to show the topographical diagnosis of the various paralyses. (Erb in *Strümpell's Textbook.*)

N.f., facial nerve; *N.p.s.*, great superficial petrosal nerve; *N.c.c.p.t.*, communicating branch with tympanic plexus; *N.st.*, stapedial nerve; *Ch.t.*, chorda tympani; *G.f.*, nerves of taste; *S.p.s.*, nerve of salivary secretion; *N.a.*, auditory nerve; *G.G.*, geniculate ganglion; *F.st.*, stylomastoid foramen; *N.a.p.*, posterior auricular nerve.

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[1] Roemer, *Augenheilkunde*, p. 700.

secretion, hearing and soft palate normal; (3) paralysis of the facial muscles, disturbance of taste and of salivary secretion, abnormally acute hearing, while the soft palate is normal; (4) paralysis of the facial muscles, disturbance of taste, diminished salivary secretion, abnormal acuteness of hearing, paralysis of the soft palate; (5) the same condi-

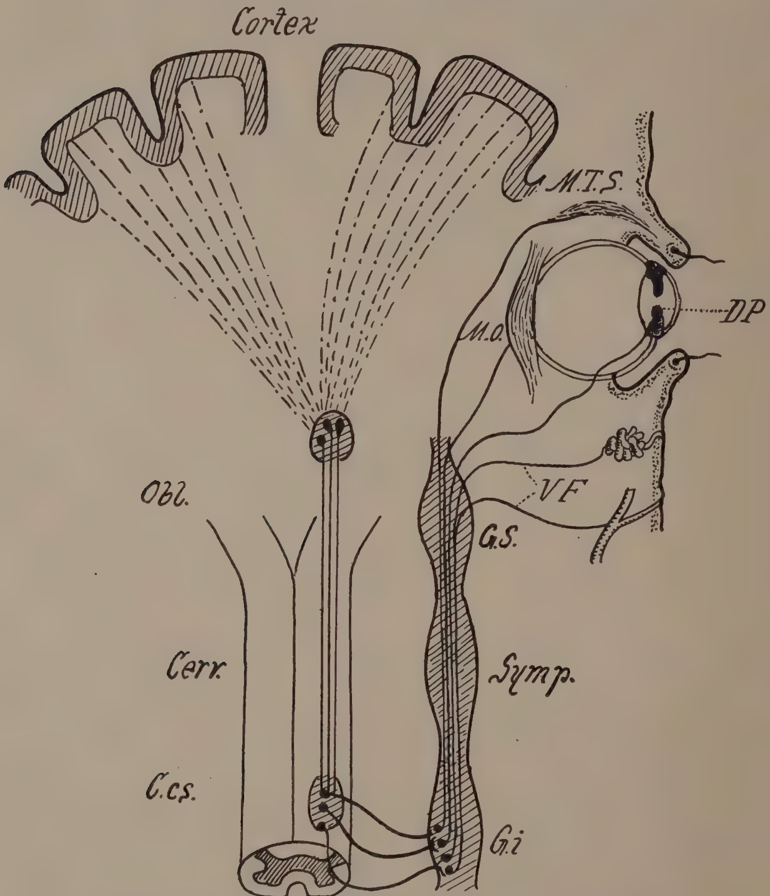

FIG. 26.—Sympathetic innervation of eyeball and orbit. (Bing, *Gehirn und Auge*, Bergmann, Wiesbaden, 1914.)

C.cs., cilio-spinal center; *Dp.*, dilator of the pupil; *Mts.*, superior tarsal muscle; *Mo.*, orbital muscle; *Gs.*, superior sympathetic ganglion; *Gi.*, inferior sympathetic ganglion; *Obl.*, oblongata; *C.rv.*, cervical cord; *VF.*, vasomotor fibres.

tions except that there is no disturbance of taste. The most frequent causes for a peripheric facial paralysis are first of all "rheumatism," second, diseases of the middle ear, and of the petrous pyramid; finally affections of the skull (fractures, tumors, and meningitis). In basal paralysis of the facial nerve, other nerves are usually involved, the VIII, VI, and V. In functional paralysis of the facial nerve (hysteria) the lower branches are usually involved while the ocular facial distribution is intact.

Sympathetic Nerve.—At the lower cervical and upper dorsal segments of the cord there is a cilio-spinal center which controls the dilatator pupillæ, the superior tarsal muscle and the musculus orbitalis. From the cilio-spinal center fibres pass through to the lowest segments of the cervical sympathetic (inferior cervical ganglion). The impulse then passes through the middle and upper cervical ganglions, and through the sympathetic plexus around the carotid artery to the orbit, where one branch surrounds the ophthalmic artery and the other represents anastomosing branches distributed to the motor and sensory nerves of the orbit, and finally arrives in the ophthalmic ganglion.

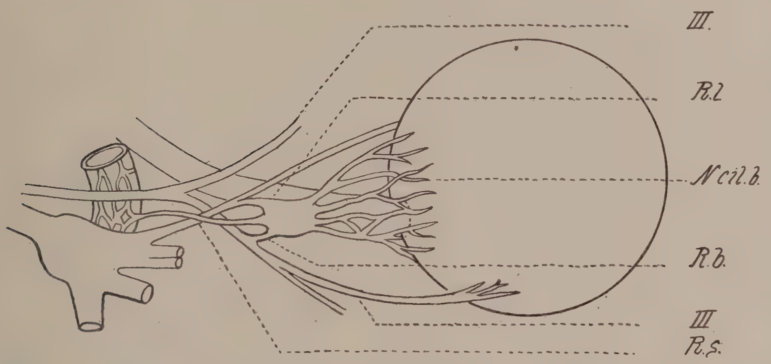

FIG. 27.—Ciliary ganglion, its roots and branches. (Poirier-Charpy-Cuneo.) *III*^{1}, superior branch oculomotor nerve; *Rl*, sensory root, nasal branch of ophthalmic nerve; *Ncb*, short ciliary nerves; *Rb*, motor root; *III*^{2}, inferior division of oculomotor nerve; *Rs*, sympathetic root.

The ophthalmic ganglion is both a cerebro-spinal and a sympathetic center situated in the orbit. It measures 2 by 1 *mm* and is situated at the external side of the optic nerve at the junction of the posterior third with the anterior two-thirds. It receives a root from the branch to the inferior oblique, another from the nasal nerve and a third from the carotid sympathetic plexus. From it arise the short ciliary nerves which penetrate the eyeball and supply the intrinsic muscles. It is of importance as a center governing the contrion of the actciliary and pupillary muscles.

Trigeminal Nerve.—The sensibility of the eye is taken care of by the V nerve. This nerve transmits sensory impressions from large cutaneous and mucous areas. In addition, it carries complex fibres which serve the sympathetic and the facial nerves. It can be divided into two neurons, a peripheric and a central one. The peripheric neuron is constituted like a spinal sensory nerve. The centripetal paths are represented by the three branches of the V nerve. The ganglion, an agglomeration of cellular centers, is the Gasserian ganglion which corresponds to the spinal ganglion of the sensory nerves. The first branch is of importance for us. Its fibres first pass into the Gasserian ganglion and then join the sensory trigeminal nucleus in the medulla oblongata and the pons. A part turns in a curved direction caudally and terminates in the substantia gelatinosa trigemini, that nuclear column which, as a direct continuation of the sensory trigeminal nucleus, extends into the upper spinal cord. It is interesting that the fibres which transmit the corneal reflex extend quite deep in the medulla oblongata near the limits of the spinal cord.

The first branch (ophthalmic nerve of Willis) is formed by the union of three important branches—nasal, frontal and lacrymal nerves. The **nasal nerve** arises at the junction of the external and internal nasal nerves. The external nasal is formed of slender filaments to the lacrymal organs, nasal filaments to the skin of the nose, and palpebro-conjunctival filaments. The internal nasal branch or ethmoidal branch, is formed by the union of the naso-lobar branch which transmits sensation from the skin of the nose and from the outer wall of the nasal cavities and by the nerve of the septum. This nerve passes from the anterior internal orbital foramen to the sphenoidal fissure, passing between the superior oblique and the internal rectus muscles, obliquely crossing the optic nerve and the ophthalmic artery. It leaves the orbit through the internal part of the sphenoidal fissure. At the ring of Zinn it comes into relation with the oculomotor nerve, the abducent nerve and the ophthalmic vein, later with the optic nerve and the ophthalmic artery. This is the only one of the sensory nerves of the orbit which travels in the interior of the muscular cone and which passes through the ring of Zinn.

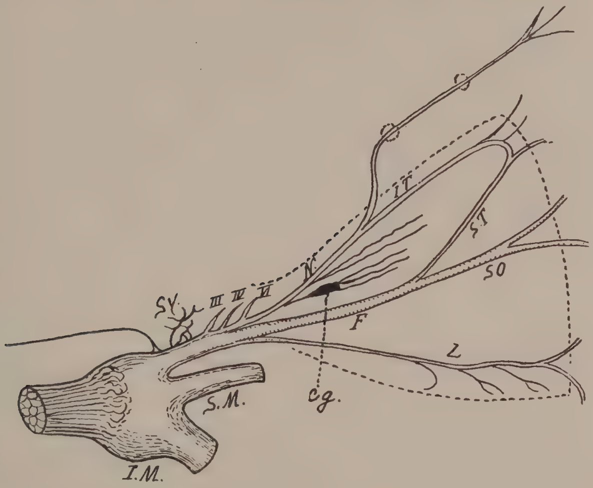

FIG. 28.—Diagram of the ophthalmic nerve. (G. D. Thane, *Quain's Anatomy.*) Dotted line is outline of orbit.

Sy., sympathetic; *Sm.*, supramaxillary; *Im.*, inframaxillary; *Cg.*, ciliary ganglion; *N.*, nasal; *It.*, infratrochlear; *St.*, supratrochlear; *F.*, frontal; *So.*, supraorbital; *L*, lacrymal.

The other two nerves, the **frontal** and **lacrymal,** are situated in the space between the muscles and the bone and pass through the sphenoidal fissure on the outside of the ring. In the orbit the nasal nerve receives the following collateral branches: the long root of the ciliary ganglion, the two long ciliary nerves, the spheno-ethmoidal branch which supplies the mucous lining of the sphenoidal and posterior ethmoidal cells.

The **frontal nerve** is the largest branch of this division. It arises from the union of the internal frontal and external frontal branches. The former is formed by branches which supply the skin of the forehead and from palpebro-conjunctival filaments. It enters the orbit to the outer side of the pulley of the superior oblique. The external frontal entering the orbit through the supraorbital notch, is formed by small filaments which supply the skin and the frontal sinus and palpebro-conjunctival filaments. The frontal nerve passes along the upper wall of the orbit above the levator palpebræ and passes through the outer portion of the sphenoidal fissure to the outer side of the ring. It is there in company with the abducens nerve to the inner side, while the lacrymal branch is to the outer side. During its passage through the orbit it receives the supra-trochlear branch, an important collateral.

The **lacrymal nerve,** which is a very thin nerve, arises from branches which supply the palpebro-conjunctival region and branches from the lacrymal gland. It travels along the outer orbital boundary above the external rectus muscle and leaves the orbit in the external part of the sphenoidal fissure to the outer side of the canal of Zinn. It receives two anastomoses, one derived from the VI nerve, the other of greater importance coming from the orbital branch of the superior maxillary nerve. The ophthalmic division is formed by the union of these three branches directly behind the sphenoidal fissure. It travels in the outer wall of the cavernous sinus in close relation to the III and IV nerves. After leaving the wall of the cavernous sinus, it forms a part of the Gasserian ganglion. During this part of its course it receives sympathetic filaments derived from the pericarotid plexus.

The **Gasserian ganglion** shaped like a crescent is lodged on the internal part of the endocranial portion of the petrous pyramid in a duplication of the dura mater. Anteriorly it receives the three branches of which it is composed. Posteriorly it continues in the trunk of the V nerve. On the inner side there is the external wall of the cavernous sinus and the nerves which are contained in this wall. Below there is the motor portion of the V nerve, the large and small superficial petrosal, the petrous pyramid where the thin bony lamella separates it from the petrous carotid canal.

Vestibular Nerve.—The vestibular nerve has its peripheric perceptive organ in the internal ear, namely, in the utriculus and sacculus and canales semicirculares. The last, the semicircular canals contain a fluid, the endolymph whose movement causes a sensory impression. Under certain conditions, when this reaches the center it causes nystagmus, certain compensatory movements of the body and the sensation of rotation. The endolymph in each semicircular canal as it moves either toward or away from the ampulla causes a nystagmus toward or away from the side of the stimulated labyrinth.

During rest there are constant stimuli passing to the vestibular ganglion and to the nuclei of the ocular muscles, which, as the stimulus is the same on the two sides, causes a state of equilibrium and brings about the vestibular tone of the ocular muscles. A similar action acting through the cerebellum causes the vestibular tone of the muscles of the body, particularly those of the trunk.

The fibres of the vestibular nerve pass from the semicircular canals to the ganglion vestibuli and then to the small-cell vestibular nucleus (nucleus vestibularis triangularis) which is situated in the floor of the fourth ventricle. The fibres which pass from this nerve directly to Deiter's nucleus are of great importance for the movements of the eyes. Deiter's nucleus is a long-drawn-out nuclear column which consists of a row of different cells. The important ones are the ventro-caudal Deiter's cells, the large cells of Deiter's, and finally, the dorso-oral cells which are in close relation to a nucleus called the nucleus angularis (nucleus Bechterew), situated in the angle of the floor of the fourth ventricle. This nucleus also contains fibres derived from the vestibular nerve. It seems very probable that these last two mentioned nuclei are related to the cerebellum, as they receive fibres from the roof nucleus. Furthermore, it is known that fibres pass from Deiter's and probably from Bechterew's nucleus to cross the middle line and to run both in an oral and caudal direction in the fasciculus posterior longitudinalis. These fibres terminate in the eye-muscle nuclei, particularly in the nucleus for the rectus externus and that part of the rectus internus nucleus which is related to lateral movements. Through the fasciculus longitudinalis posterior the eye muscles are influenced by Deiter's nucleus (labyrinth) and partly by the cerebellum. The fibres to the nuclei of the ocular muscles bring about the vestibular ocular movements. The fibres to the cerebellum and the spinal cord cause the vestibular tone of the muscles of the body, while the fibres to the cortex give the sensation of turning.

The *vestibular apparatus* can be examined by testing the vestibular ocular movements, the vestibular movements of the body at large, the sensation of turning and other associated symptoms.

VESTIBULAR NYSTAGMUS which Uhthoff calls nystagmus-like contractions, is composed of a rapid and a slow phase. Bárány[1] has shown that the slow movement is vestibular in origin and that the rapid movement is a central, supranuclear one. In describing nystagmus its direction is usually determined by observing the rapid phase. Vestibular nystagmus occurs in a number of forms—horizontal, rotatory, vertical, etc. It is increased when the eyes are turned in the direction of the rapid movement, while it is diminished or arrested when the eyes are turned in the opposite direction.

In many normal persons the eyes show slight nystagmus-like movements in the terminal lateral positions. When this nystagmus is marked it is pathological, while every form of nystagmus with the eyes in the primary position, even if slight, is pathological. Bárány finds that in examining for nystagmus it is desirable to rule out the attempt of the eyes to fix an object, and recommends opaque glasses; then under certain conditions a nystagmus will become visible.

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[1] Bárány, Handbuch d. Neurologie, Lewandowsky, Vol. 2, p. 919.

In diseases of the peripheric end organ the spontaneous nystagmus is a combination of a horizontal and rotatory nystagmus, while every other form of spontaneous nystagmus is intracranial.

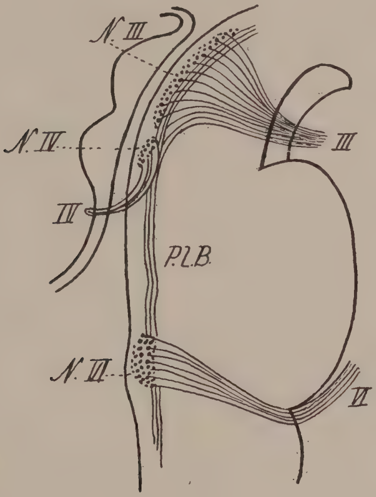

Fig. 29.—Diagram of the origin of the *III*, *IV*, and *VI* nerves. (Modified from Gowers, *Quain's Anatomy*.)

The paths by which voluntary movements of the eye muscles are effected are not known. At present an isolated voluntary tract only has been accepted for the case of the levator palpebræ. All other movements are associated and their center lies at the base of the second frontal convolution. The fibres run from this area together with the pyramidal tract in the internal capsule and then in the pes pedunculi. They then separate from the other fibres at the anterior extremity of the pons and travel dorsally in the region of the fasciculus longitudinalis posterior after they have crossed to the other side. In the fasciculus longitudinalis posterior, orally from the abducent nucleus, the fibres pass on to their final termination in the nuclei. In considering associated lateral movements of the eyes, it is not necessary to assume a special center for their development. Paralysis of associated ocular movement results from a lesion in the cortex or in the course of the tract directly in front of its bifurcation, just orally from the abducent nucleus. As this path crosses over to the other side, a paralysis of the associated lateral muscles in cortical lesions must be a crossed one; when the lesion is in the region of the abducent nucleus, the paralysis is of the same side.

The associated lateral movements are furthermore influenced from still another direction, namely, from Deiter's nucleus. Its lesion leads to nystagmus, that is, ryhthmic nystagmus, which is composed of a rapid and a slow component. A lesion of the ventro-caudal Deiter's causes horizontal nystagmus, and a lesion of the oral portion of the nucleus produces vertical nystagmus. Hertwig-Magendie's strabismus (vertical strabismus) in lesions of the cerebellar peduncles is possibly related to Deiter's nucleus. A lesion of Deiter's nucleus can produce in addition to nystagmus a paralysis of associated ocular movements, or at least a turning of the eyes to one side.

There are a number of ways of studying the **vestibular reactions** on the part of the ocular muscles. The most important one and quite sufficient for the ophthalmologist is the caloric test of Bárány. On irrigating the right ear of a patient in an upright position with water below body temperature a horizontal nystagmus to the left side sets in after about twenty seconds and continues for two minutes. If the temperature of the water is higher than that of the body a rotatory nystagmus results toward the right side.

In other words, the movement of the endolymph varies with the temperature of the water, as it is increased or decreased. The temperature of the water when it is to be below body temperature should be between 25° or 30°C.

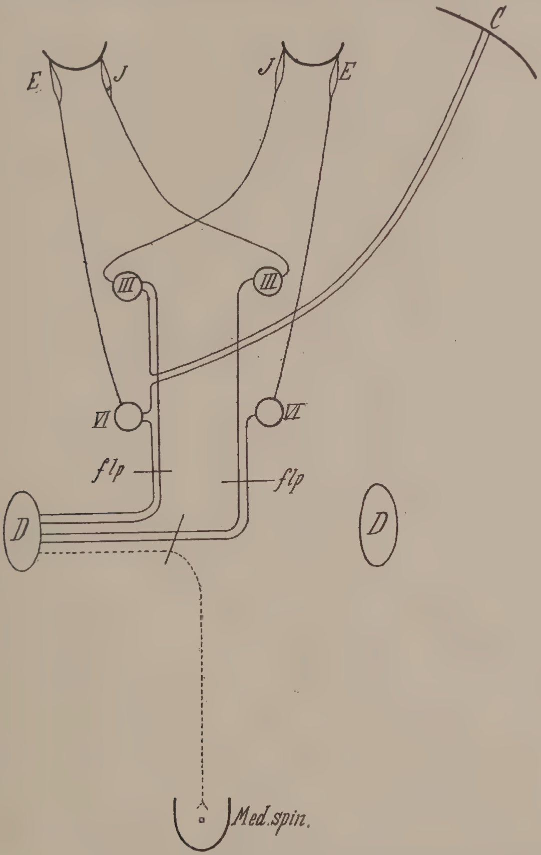

FIG. 30.—Diagram to explain nystagmus.

E, external rectus; *I*, internal rectus; *III*, oculomotor nerve; *VI*, abducent nerve; *Flp*, posterior longitudinal bundle; *D.*, Deiter's nucleus; *C.*, cortex; *Med. spin.*, medulla spinalis.

During irrigation the eyes should be carefully observed and as soon as nystagmus sets in, the irrigations must be arrested, to avoid vertigo, nausea and vomiting. If no reaction results from 25°C., colder water can be used. To irrigate with hot water a temperature from 45° to 48°C. is advisable. Generally it is sufficient to use cold water to make a diagnosis. The test becomes difficult if spontaneous nystagmus is already present because it is hard to tell whether the movements are augmented or diminished. In the unconscious this caloric reaction can also be practised; instead of a nystagmus the eyes will deviate to the side which has been irrigated with the cold water. If the peripheric vestibular apparatus is disturbed or the vestibular nerve is paralyzed irrigation with cold water will produce no reaction whatever.

The vestibular apparatus not only influences the muscles of the body but also those of the extremities. On producing horizontal nystagmus to the right side, the attempt of the patient to point with closed eyes always shows a variation inasmuch as the past pointing takes place to the left side. If the head is bent toward the right shoulder the past pointing takes place above the object.

The vestibular ocular movements have been studied in paralysis of ocular muscles and in paralysis of associated movements, to determine whether the condition is due to a peripheric (nuclear) lesion or not. In the case of a nuclear lesion the vestibular stimulus produces no increase in the movement along the direction of the paralysis, while vestibular nystagmus results normally in any direction which is not affected by the paralysis. In paralysis of the associated movements which are supranuclear a different condition exists.

Bárány has shown that the slow movement of the nystagmus originates from the end organ. The rapid movement has a central origin arising in the supranuclear center. Assuming a focus in the left supranuclear center the associated movement of both eyes to the left is paralyzed. The rapid movement of the horizontal nystagmus to the left side is also affected. On stimulation, in place of the horizontal nystagmus toward the left, both eyes now become deviated to the right and remain in this position. Bielschowsky and Steinert[1] have observed cases of paralysis of vertical associated movement in which the vestibular motility has been preserved. In recovery from a paralysis of associated movement a marked nystagmus to the side of the paralysis always appears. In the supranuclear paralysis the absence of vertigo is noticeable.

THE RÔLE OF THE EYES IN ORIENTATION AND EQUILIBRATION.—Among the nervous impulses which act in preserving our equilibrium, those obtained from the eyes are important, because the knowledge of the relation of external objects to the body gained from the contraction (*i.e.*, innervation) of the eye-ball muscles is one of the most important sources of guidance to the centers that regulate the muscular contractions for maintaining bodily equilibrium. In addition to this independent service there is a close association between the eyes and the static labyrinth, which, however, is not directly connected with equilibrium, but has a different purpose, as is shown in the following:

The posterior longitudinal bundle which connects the various nuclei of the ocular muscles is in close relationship with the vestibular nerve. The vestibular nerve thereby is closely associated with the ocular muscles, just as it is with all the muscles of the body. It serves the purpose of maintaining the muscles in a state of tonic contraction. If this nerve be divided on one side, it will result in a complete loss of tone of the muscles of the body of the opposite side and in nystagmus. The vestibular apparatus is also in close relationship with the cerebellum, by means of Deiter's nucleus, so that the cerebellum is a further active factor in the regulation of our ocular movements. Bing[2] calls the cerebellum a reflex apparatus serving for the preservation of equilibrium, not only for the muscles of the body, but for the eyes. It received centripetal impulses from the static labyrinth and from the ocular muscles by means of Deiter's nucleus, which assist in its regulating activity. On the other hand, it sends forth centrifugal impulses through this same nucleus, which preserve the position of equilibrium and control the ocular movements.

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[1] Bielschowsky and Steinert, Muenchener med. Wochenschrift, 1906, 33 and 34, quoted from Bárány.

[2] Bing, Gehirn und Auge, Bergmann, Wiesbaden, 1914.

In certain animals the position of the eyes and their movements are governed nearly exclusively by the ear. The eyes in these animals, if the head is not moved, are practically motionless. As the head moves, the eyes make a compensatory movement in the opposite direction which originates in the labyrinth. There is a constant action of the labyrinth on the ocular muscles. It holds the ocular muscles in check, giving them a definite tone, so that a lesion of one labyrinth causes a preponderance of the action of the opposite one. In man this association between the eyes and the labyrinth also works for producing compensatory movements, though these are not as important as in the lower animals. The purposes of these compensatory movements according to Sherrington, are presumably for maintaining the normal relation of the eyes to the horizontal. These movements, Wilson and Pike have shown, are a direct reflex response, independent of the cerebellum, to insure that the retinal images shall not be disturbed by each movement of the head. It is well-known that if both labyrinths are destroyed, these compensatory movements are abolished.

Conjugate deviation can be regarded both as a symptom of irritation and as a symptom of defect, the latter when there is a secondary contracture of the non-paralyzed lateral turning muscles of the eye; the former a symptom of irritation when there is no paralysis of associated ocular motion and when the fibres for the associated lateral movements are in a state of irritation through the presence of a lesion. Conjugate deviation is usually only of short duration and occurs in lesions situated in the frontal lobe (base of the second frontal convolution, at the gyrus angularis, and in the pons). The deviation turns to the opposite side of the paralyzed associated ocular movement. In right-sided cortical lesions the deviation is to the right; when the focus is in the pons, the deviation is to the left. A number of cases have been observed in which a conjugate deviation was not dependent upon a lesion of the nervous paths governing ocular movement, but was due to a disturbance in the system of Deiter's nucleus. In this case the eyes may remain deviated in one direction for a long time, though the laws governing this disturbance have not been discovered.

The **superior or cortical centers** for ocular movements are not definitely understood but are described as two—one a positive or visuo-motor which is placed in the region of the visual center on the internal surface of the occipital lobe, the other or anterior, supposed to be situated at the foot of the second frontal convolution and the angular gyrus. The former center governs the ocular movements associated with visual sensation; the second the movements associated with general sensory impression. Mott and Schäfer[1] have shown that irritation of the anterior center in its middle part causes lateral movements of the eyes; in its upper part, lateral movements with depression; and in its inferior part, lateral movements with elevation.

Some authors assume intermediate **coördinative centers** (supranuclear) for binocular parallel movements of the eyes. The coördinative centers have two functions: first, to coördinate the movements of the two eyes for binocular and for the movements of the eye lids, as well as preserving the equilibrium and general orientation. The center for the coördination of the binocular movement with parallel axes are two in number, right and left. Their place is not exactly understood, though it is probable that they are located in the pons. The center of the coördination without parallelism (convergence) is also situated in this region, but is not exactly localized.

The center for the movement of the eye lids has been located at the level of the upper margin of the pons. The centers associating ocular movements with the sense of orientation and equilibrium are closely related to the central connection of the vestibular apparatus and they are placed in Deiter's nucleus. The cerebellum has an important function in preserving coördination.

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[1] Quoted from de Lapersonne et Cautonnet, l. c., p. 20.

The peripheric centers are closely united by fibres belonging to the posterior longitudinal bundle which connects the III, IV, and VI nerve nuclei on each side.

The cortical and peripheric centers are associated by fibres which converge in the direction of the internal capsule, grouped in two bundles. The anterior from the sensory-motor center travels with the fibres from the frontal lobe. The posterior fibres from the visuo-motor center of visual sphere are contained in the optic radiation. In the internal capsule the anterior bundle passes to the posterior part of the anterior arm and the posterior bundle occupies the posterior-inferior part of the optic thalamus. The motor ocular fibres then travel in the dorsal part of the peduncle and of the pons to decussate and cross the median line at the upper level of the pons, and finally to be distributed to the peripheric nuclei with the possible intervention of a supranuclear or coördinate center.

THE PUPIL

In the **examination of the pupil** we must pay attention first to its size, its form and its reaction. The normal size of the pupil is from 3 to 5 *mm*. This figure holds only for adults. In children, in anæmic persons it is larger, in old people it is smaller. If the pupil is contracted to 1 to 1½ *mm*, we speak of miosis; if the pupil is larger than 5 *mm*, the condition is described as mydriasis. Normally the size of the pupil in both eyes is the same. In nervous, excitable persons a small difference in illumination produces an apparent difference in the size of the pupil. This, however, is promptly equalized when the illumination is uniform. The form of the pupil is of the greatest importance. It is usually circular, though it may normally be somewhat oval or elliptical; in any case, the margin is usually entirely uniform. Asymmetry of the margin, a slightly displaced pupil, an irregular margin, are always to be regarded as pathologic (congenital change, iritis, lues).

There are two physiologically different forms of pupillary movement: I. Reflex pupillary movement, which produces a contraction or dilatation of the pupil on changes in the illumination, on sensory stimuli, or psychic influences; II. Associated movements of the pupil. The pupillary fibres are stimulated together with other actions of the oculomotor or one of the accessory nerves (facial, trigeminal).

Reflex pupillary movement is the most important of the various reactions of the pupil. There is a direct and a consensual reaction occurring in the same way, at the same time and to the same degree. It is to be remembered that sight and degree of light reaction are not always equal. Good pupillary reaction has been observed in blindness following choked disc or central optic atrophy. As has been previously stated, the anterior corpora quadrigemina represent a reflex organ which transmits the light reflex to the motor paths. The most important clinical feature of these reflexes is the pupillary reflex, and particularly the light reflex of the pupil. There is to-day no question but that the centripetal fibres of this reflex arc run in the optic nerve, as Karplus and Kreidl were able to produce changes of the pupils by irritation of the optic nerve. The way, however, by which these optic nerve fibres pass to the motor nuclei of the pupil is not definitely understood. According to Bernheimer, the fibres pass through the brachium of the anterior corpora quadrigemina before reaching this structure; they then pass around the Sylvian aqueduct in a curved direction to the oculomotor nucleus. In any event, to insure the light reflex, the optic nerve must be intact on the one hand and the sphincter nucleus must be normal on the other, therefore in loss of the light reflex, as Bach

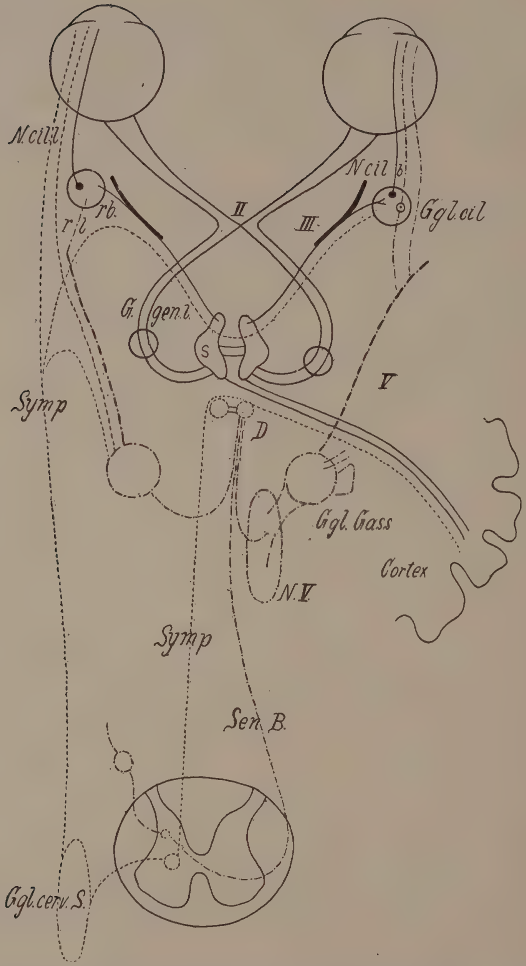

FIG. 31.—The nervous mechanism of the pupillary reaction. (After Marburg, *Die diagnostische Bedeutung der Pupillenreactionen*, Wiener Klinik, 1903.) Pupillary reflex tracts.

D., dilator nucleus of oculomotor nerve; *G.gl.*, Gasserian ganglion; *N.V.*, nucleus V. nerve; *Sens. B*, sensory tract except head supply from *V*. Cortex. *Symp.*, sympathetic tract; *G.gl.cerv.s.*, superior cervical sympathetic ganglion; *N.cil.b.*, nervi ciliares breves; *N.cil.l.*, nervi ciliares longi; *Ggl. cil*, ganglion ciliare; re, radix longa, ciliary ganglia; *rb.*, radix brevis, ciliary ganglia; *II*, chiasm; *III*, oculomotor nerve; *V*, trigeminal nerve; *G.gen.l.*, external geniculate body; *S.*, sphincter nucleus.

suggests, the place where the light stimulus is transmitted to the motor nucleus alone must have suffered. There is no proof for any of the other theories to explain this question.

Sensory or sympathetic reactions of the pupil consist in a dilatation of the pupil, following sensory or psychical stimuli. Every intensive psychical process and every sensory stimulus is associated with dilatation of the pupil even when it is preceded by a voluntary impulse or by imagination. This reflex was formerly described under a variety of different names.

(*a*) The so-called sympathetic reflex, the one produced by all forms of sensory stimuli and irritants of the skin and of the higher sensory organs (pin pricks, noises, odors, intense light).

(*b*) Psycho-reflex is the dilatation of the pupil in mental work, close attention, muscular contraction or the mental state of fright, anger, fear.

(*c*) Pupillary cortical reflex, which is the reflex produced by the imagination of a bright or dark object. Haab stated that the imagination of a dark object leads to a pupillary contraction, an observation which has not been confirmed, especially if simultaneous convergence and ocular fixation are excluded.

Psycho-reflex pupillary dilatation shows individual variations, being more pronounced in women though it is not absent in health, except in the new-born.

Recent investigations have shown a distinct relationship between the contraction of the voluntary muscles and certain pupillary changes. Redlich has obtained a dilatation of the pupil in normal persons after heavy muscular work. The so-called paradoxical pupillary reaction (dilatation of the pupil on exposure to light) is probably a sensory reaction.

The sympathetic pupillary reaction suffers whenever there is a disturbance in the ocular sympathetic. The dilatator of the pupil is paralyzed, the sphincter contracts and miosis is present. This miosis is frequently associated with sympathetic ptosis and enophthalmos following paralysis of the smooth muscles of the eye (Horner's symptom-complex, sympathetic ophthalmoplegia Breuer-Marburg). The center for this disturbance must be situated in the angular gyrus of the parietal lobe. The paths travel from this center in the posterior segment of the interior capsule to the region of the corpora quadrigemina, where the fibres decussate. They then continue in the medulla oblongata, attain the spinal cord, leaving at about the sixth to the eighth cervical segment (centrum ciliospinale, Budge's center) to reach the cervical sympathetic and through this pass to the eye. The dilatator nucleus is probably situated in the vicinity of the sphincter nucleus, though Karplus and Kreidl believe that there is a sympathetic center in the regio subthalamica. In the eye itself there is a relay station in the ciliary ganglion which contains sensory fibres from the ciliary nerves (trigeminus) in addition to motor and sympathetic cells.

While irritation of the sympathetic nerve causes dilatation of the pupil, enlarged lid opening from retraction of lids and slight exophthalmos, instillation of cocaine in the eye produces the same signs and an insufficient accompanying of the upper lid on looking down (v. Graefe sign).

The Associated Movements of the Pupil.—The associated movements of the pupil are always pupillary contractions and occur with the simultaneous innervation of other nerves, especially of the oculomotor, facial and trigeminal nerves. They are of less practical importance than the preceding.

(*a*) Convergence.—An association between the sphincter of the iris with the internal rectus muscles.

(*b*) Accommodation.—This is an association of the sphincter of the pupil with the ciliary muscle. As the accommodation is always associated with convergence, we have associated action of these three branches of the III nerve, sphincter of the pupil, ciliary muscle and internal rectus. The central tract of the convergence reaction is not known. The nucleus of the ciliary muscle is probably situated in the anterior lateral nuclear region of the III nerve, somewhat posterior to the sphincter nucleus. The single median nucleus in the oculomotor region is regarded as the center for accommodation. Its center is probably situated in the median nucleus of the oculomotor root. A deviation of this reaction from the normal is described as myotonic. The pupil then remains after contraction for a certain brief time in this condition, and then gradually dilates instead of dilating suddenly.

(*c*) The lid closure reaction of the pupil (orbicularis phenomenon, v. Graefe, Westphal-Pilz reaction) consists in a contraction of the pupil on the attempted closure of the lid. It is an associated movement of the iris with the orbicularis, a synergia of the oculomotor and facial nerves. Every centrally caused closure of the lid is associated with a narrowing of the pupil, though the latter is usually neutralized by other pupillary movements. There is no clinical importance to this phenomenon. A synergic action with the abducent nerve has been occasionally observed in the form of a narrowing of the pupil. This has been explained by the presence of pupil contracting fibres in the abducent nerve.

PUPILOMOTOR AREA IN RETINA.—It was formerly believed that the entire retina was capable of producing a pupillary reaction. Hess has, however, shown that only a very small area is pupilomotor. This area consists of a small central part of the retina, about 3 *mm*, surrounding the macula. We can, however, not separately illuminate the central and excentric parts of the retina. In all clinical examinations the diffuse light is not negligible; hence the clinical examination of the pupil is always an imperfect one.

EXAMINATION OF THE PUPIL REACTION.—The procedure of studying the pupil reaction consists in the determination of the size of the pupil in: (1) reduced illumination; (2) strong binocular illumination; (3) strong monocular illumination; (4) on convergence. The examination is made in a dark room, the lamp being behind the head of the patient, whose gaze is directed toward the distance. With the aid of the ophthalmoscope mirror light is thrown into the pupils at a distance of 40 *cm*. The size of the pupils can then be noted. The direct and indirect reaction to light is observed by concentrating the rays of light from the light source situated somewhat in front and to the side of the patient. The light is concentrated with a convex lens and the eye is illuminated. The convergence reaction is studied by asking the patient suddenly to fix the finger held directly in front of him.

Diagnostic Importance of the Pupillary Reaction.—The normal reaction of the pupil to light varies in extent, depending upon the size of the pupil, and in rapidity. Abnormal reactions of the pupil may furnish information for topical diagnosis. In a lesion anterior of the chiasm the direct reaction of the eye on the side of the lesion and the consensual reaction of the other eye are lost. The disturbance of vision and the pupillary reaction are not necessarily related. In retrobulbar neuritis where the sight is so much affected, the pupillary reaction remains. A lesion in the chiasm or in the optic tract gives, in addition to hemianopsia, the hemiopic pupillary reaction. The hemiopic pupillary reaction without hemianopsia indicates a lesion between the primary optic centers (the anterior quadrigeminal body and the III nerve nucleus). This represents the last stretch of the afferent reflex pupil arc. The most important change in the pupil is **reflex iridoplegia** (Argyll-Robertson pupil), in which there is a loss of direct consensual mobility. The reflex center is cut off from all afferent reflex fibres and the lesion must be near the center (sphincter nucleus). This condition occurs either as a permanent one or a transient one. (*a*) Permanent always indicates severe organic nerve disease and points to positive syphilitic antecedents and may be followed by tabes, paralysis or cerebral syphilis. (*b*)

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Transient occurs exceptionally in neurosis, sometimes after trauma, meningitis, typhoid fever, influenza, pneumonia and diphtheria; also in intoxications from atropin, chloroform, filix mas, mushrooms and ptomaine poisoning, and especially alcoholism when it is due to a polioencephalitis in the III nerve nuclear region. Finally, it occurs in attacks of neurosis, epilepsy and hysteria. The **associated movements are of secondary diagnostic importance** as they occur only in connection with other movements upon which they depend. Marburg suggests that they had better be called phenomena. These phenomena are absent when the sphincter nucleus or the afferent III nerve fibres are affected. Permanent accommodative immobility is due to tabes, paralysis, lues, infectious fevers, atropin and diabetes.

The absence of the light reaction of the pupil suggests first of all progressive paralysis, second, tabes. Ten per cent. of the cases of reflex iridoplegia are caused by cerebral lues. Other cerebral affections are: tumor 6 per cent., multiple sclerosis 1 per cent., injuries to the head and spinal cord 2 per cent., tobacco and alcohol poisoning 1 per cent. The condition may be one-sided or bilateral. Both pupils may be totally paretic (both to light and to accommodation); this condition occurs less frequently than reflex iridoplegia in tabes, and progressive paralysis, and its occurrence increases in the various forms of cerebral lues. The convergence reaction can be elicited even if the miosis is well developed.

In every **miosis** and **mydriasis** and in every irregularity of the pupils the accommodation must be examined. If one pupil is dilated and the accommodation involved, internal ophthalmoplegia is present; this is usually a central lesion. Slight degrees of anisocoria are not infrequent; if the accommodation and the various pupillary reactions are normal, it is without significance.

Coppez says that the pupils should be examined by both bright and diminished light; in a bright light a lesion of the dilator may not be perceived because that muscle is not so powerful as the sphincter. If the pupils are unequal instill 5 per cent. cocain into the eye whose pupil is the larger; there is no supplementary dilatation the mydriasis is due to excitation of the dilator fibres; it is due to paralysis of the III nerve if the supplementary dilatation is so great that the iris becomes almost invisible. If the supplementary dilatation is moderate (1 to 2 *mm*) the pupil is normal. Then a drop of the same solution is put into the other eye; if dilatation is nil or very feeble, we have a miosis from paralysis of the dilator fibres; if the dilatation is moderate (1 to 2 *mm*) the pupil is normal. A spasmodic miosis is not affected by cocain. Now put a drop of 1 per cent. atropin into the eye with the contracted pupil; if this produces only a slight dilatation the miosis depends upon paralysis of the sympathetic. This is the case in the contracted pupils of tabetics. If a marked dilatation is produced by atropin, the contraction is due to spasm of the sphincter. According to Gautrelet[1] cocaine dilates the pupil if the nerve terminals in the dilator muscle are intact. Adrenalin causes a mydriasis only in cases of miosis following a lesion of the sympathetic nerve. A few drops of a 1:1000 solution causes a dilatation of such a narrow pupil after a few minutes, while a normal or a spastically contracted iris remains unchanged.

The so-called HEMIOPIC PUPILLARY REACTION, which has been defined by Hess as "hemikenesia," is of importance to diagnose the exact seat of a lesion in the optic paths. The possibility of exactly determining the presence of this reaction has been questioned by many. One of the most important sources of error, as shown by Hess, is the reflected light which can not be controlled. He has particularly drawn attention to the fact that light through the sclera is capable of producing pupillary movements. As these factors can not be excluded, Hess has neutralized them by making their degree constant. He has devised an apparatus[1] based on the principle of alternating illumination by which the amount of light is always constant. This principle of alternating illumination depends upon two points of illumination whose intensity is changed so that at one time the seeing half of the retina is stimulated while at another time the blind half is stimulated, but the total amount of light entering the eye does not show any change. Thus the disturbances which were observed in previous methods, due to diffuse light, are corrected. In making this test, it is first of all necessary to determine the amount of light which the two halves of the normal eye demand, in order to cause, on alternating illumination, the pupils to remain of the same size. This has to be separately tested for each eye.

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[1] Gautrelet, Archives d'ophthal., April, 1909.

In 1889 Wilbrand suggested a method of examination which he called the **hemiopic prism phenomenon.** Wilbrand found in cases of homonymous hemianopsia where the autopsy subsequently showed the lesion to be situated posteriorly to the primary optic centers of the eye, that the eyes made a prompt and equalizing movement when the image of an object was thrown by means of a prism on the blind half of the retina. This compensatory movement was found absent in a case of basal meningitis. Wilbrand recommends the test to be made as follows: The patient is seated in front of a blackboard and his attention is fixed upon a small piece of white paper. The other eye is bandaged. Suddenly a strong prism is so placed before the eye that the patient no longer sees the object. It is then necessary to observe whether the eye makes a compensatory movement in the direction of the axis of the prism and whether the movement in the opposite direction is performed when the prism is then again rapidly removed. It is necessary to introduce the prism very quickly, so that the patient is not able to follow the path of the image.

Behr, in 1909, in an article on the "Topical Diagnosis of Hemianopsia," confirms the value of this prism test and believes it to be a very much more exact method of examination than the one usually practised to determine the hemianopic pupillary reaction. Bielschowsky, however, believes that this compensatory movement of the eye is the result of the experience gained by the patient. Jess[1] has been able to examine eight cases of hemianopsia, both as to the hemikinesia and the presence of Wilbrand's compensatory movements of the eyes. The cases examined exhibited lesions in the chiasm, in the tract and in the occipital lobe. In the cases of chiasmal and tract lesions a hemikinesia was positively present. In the case of central lesion the pupil was absolutely undisturbed. In the other five cases the symptoms pointed to central hemianopsia. In all of these the reflex compensatory movements of the eye were absent as well as in the first two with peripheric lesions.

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[1] Hess, Arch. f. Augen'lk., Vols. 58 and 60.

Clinical examination has confirmed that the Wilbrand prism test can not be made useful in the topical diagnosis of hemianopsia. The examination of the hemiopic pupil, however, is possible by means of the Hess apparatus, and in these cases it proved to be a definite diagnostic means for localizing cerebral affections.

THE CEREBRO-SPINAL FLUID

The cerebro-spinal fluid circulates in a definite direction, passing from the third ventricle into the subarachnoid space. The pia is closely adherent to the brain surface, while the arachnoid travels from one cerebral convolution to another. The interspace is the subarachnoid space which is traversed by fibrous trabeculæ. The size of this space varies; on the convexity of the brain it is generally only a narrow fissure.

In certain situations the two lining membranes separate to form large cavities which are known as cisterns. The largest cistern (cisterna magna) occupies the interval between the medulla and the lower surface of the cerebellum.

[1] Jess, Archiv f. Augen'lk., Vol. 71, p. 66.

The cisterna pontis is situated at a corresponding position anterior to the pons. These two cisterns form a water bed, so to speak, for the brain to rest upon. Somewhat further upward there is another space formed by the deep hollow between the projecting temporal lobes, known as the cisterna basalis, which contains the large arteries forming the circle of Willis. This cisterna basalis is in communication with wide subarachnoid channels passing into the Sylvian fissure and into a space surrounding the optic chiasm from which they are then prolonged into the great longitudinal fissure.

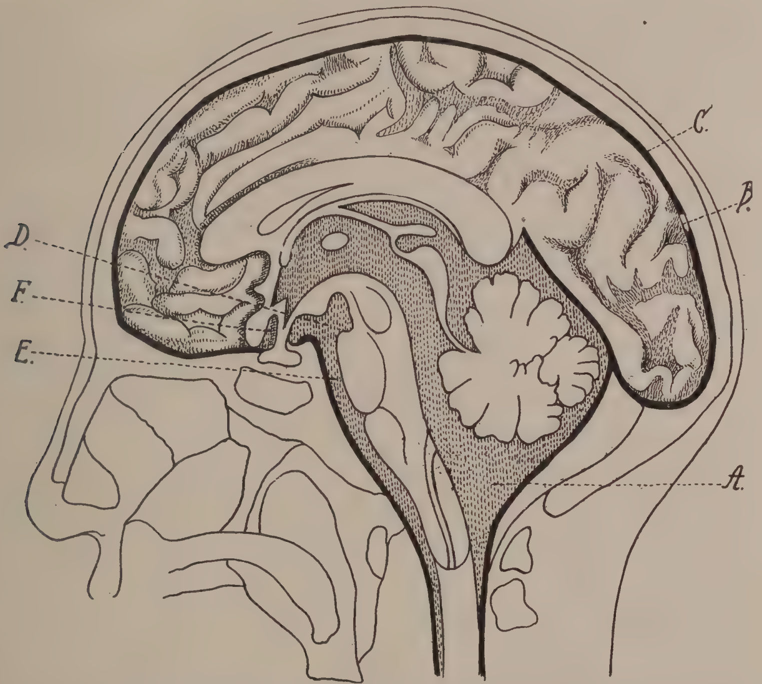

Fig. 32.—The sub-arachnoid spaces and cisterns. (From Merkel, modified by Quincke.)

A., Sub-arachnoid space cisterna magna; *B*, sub-dural space; *C*, dura; *D*, cisterna basalis; *E*, cisterna pontis; *F*, cisterna anterior to optic chiasm.

The chief origin of cerebro-spinal fluid is the choroid plexus, called the choroid gland by Mott, which forms a protrusion of the arachnoid and of the pia into the III ventricle. The normal liquor is no longer regarded as a transudate of the blood, but is now believed to be a secretion. The absorption of the liquor takes place principally through the veins situated on the surface of the brain and the nerve sheaths. Its function is probably to insure a mechanical protection to the brain centers, thereby regulating the circulation in the brain.[1]

The cerebro-spinal fluid is clear, colorless, and faintly alkaline, with a specific gravity of 1006 to 1008. It contains a trace of globulin and a substance which reduces Fehling's solution, probably glucose, and an occasional lymphocyte. The cerebro-spinal fluid has the same constituency as the perilymph of the internal ear and the aqueous humor. The greatest amount of the dry substances is furnished by sodium chloride. Together with the other members of this group, it contains the smallest amount of albumen of any of the body fluids. The liquor is an excellent agent to examine for pathologic changes in the nervous system, because the amount of formed substances is small. Under normal conditions there are only a few substances which pass from the blood into the subarachnoid space whose number and quantity change remarkably the minute there is disease of the central nervous system.

If the fluid is turbid, this indicates the presence of polynuclear leucocytes, usually accompanying acute inflammation of the meninges. A large number of lymphocytes may be present without causing turbidity, so that in tuberculous meningitis, though many mononuclear cells may be present, the fluid still remains clear. The presence of microörganisms, furthermore, need not produce a turbid fluid. Its color changes but rarely. In hemorrhages of the brain and spinal cord it is frequently hemorrhagic for a few days only. The presence of fibrinous coagula in the liquor on standing is not a specific criterion for tuberculosis, as it has been found by Reichmann in hydrocephalus and in other chronic diseases.

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[1] Reichmann, M. m. W., p. 468, 1912.

Of greatest importance clinically is the determination of albumen and of cellular contents. The quantity of albumen is always found increased in paresis and tabes, and to a lesser extent in cerebral lues. The number and nature of the cells present in the fluid are of great importance. To determine the cytology, the liquor is centrifuged, a given quantity of the deposit is spread on a cover glass, dried, stained and examined under the microscope in a counting chamber. In all cases where there is no pathological process present the number of cells found is an unusually small one, only two or three lymphocytes in a field. Pathologically significant is an increase of the cell count of more than ten pro cubic centimeter. In tabes, according to Reichmann, the number of cells varies from between 20 to 219. In purulent meningitis, over a thousand may be found in a cubic centimeter. In two cases of tumor, notwithstanding increased albumen, lymphocytosis was found absent. This has been found in other similar cases, and shows that in the presence of increased albumen, if there is no pleocytosis, the presence of a spinal tumor is probable. Generally speaking, in acute inflammatory processes, especially in meningitis, the polynuclear varieties preponderate, except in the case of tubercular meningitis. When the acute symptoms disappear the polynucleosis is gradually transformed into a lymphocytosis of a less degree. Lymphocytosis is found present in all chronic diseases of the brain and of the spinal cord, and particularly in the chronic affections due to syphilis and tuberculosis. In addition, certain mental diseases are accompanied by an increase in the lymphocytes. In tuberculous meningitis lymphocytosis is the rule, but in some cases a considerable proportion of the cells may be polynuclear. Demonstration of tubercle bacilli in the coagulum, which usually forms when the fluid is allowed to stand, will settle the diagnosis.

The earliest and most certain physical sign of general paralysis and tabes is a lymphocytosis in the cerebro-spinal fluid. The earlier the case the more marked is the lymphocytosis. The presence of lymphocytosis together with a positive Wassermann reaction in the cerebro-spinal fluid, in the opinion of Boyd,[1] is worth all of the other physical signs put together. In tertiary syphilis of the nervous system lymphocytosis usually occurs, but it is not nearly so marked as in general paralysis and tabes.

Lumbar puncture, if proper directions are followed, is practically free from danger, especially if the patient remains in bed after the puncture. Contra-indications are brain tumors which constitute the dangerous cases. About 30 fatal cases are on record. Whenever the pressure is very much increased, care must be taken. Normally the liquor runs off in drops. A pressure of 120 when the patient is lying down can go up to 300 when the patient assumes a sitting position. In order to get satisfactory figures, it is well to wait until the patient has quieted down before reading off the height of the pressure. In pathological conditions the pressure may rise to over a thousand. In these cases, of course, it is necessary to be cautious in allowing it to escape. The normal pressure varies between 60 and 120: 120 to 180 is the margin; 200 and over is pathologic. In children the pressure is one-third lower. It varies regularly with the heart action and with the respiration.

Increased intracranial pressure is in most central diseases dependent upon increased function of the secreting epithelium and retarded absorption as a result of angioneurotic, infectious or toxic causes. This increased pressure in turn acts unfavorably on the functions and nutrition of the central nervous system. The clinical symptoms are not necessarily in proportion to the degree of increased pressure. As a rule the symptoms in acute rise of pressure are more marked than in the chronic. Pathologically increased lumbar pressure is found in many pathological conditions, in congenital as well as in acquired hydrocephalus. One of the most important signs of a preceding injury of the skull is increased lumbar pressure. Nearly all organic diseases of the brain and of the spinal cord show increased lumbar pressure, particularly metasyphilitic diseases, tumors, arteriosclerosis. It is natural that all forms of infectious meningitis can produce increased lumbar pressure, which may rise during the process of healing. Increased pressure is found in cases of liquor congestion, in acute infectious diseases, in cerebral edema, chronic nephritis, chlorosis, cortical apoplexy and hemorrhages. Acute intoxications, alcohol, uremia, can also increase the liquor pressure. Many functional mental diseases may also cause increased pressure. An increase in the amount of liquor seems only present when the pressure is very much increased. General conditions of weakness and low blood pressure cause a reduced lumbar pressure. A fall of the lumbar pressure through too large a removal of liquor can be followed with the clinical symptom of meningism: vertigo, nausea, rigidity of neck, headache. This results rather from the amount of fluid abstracted than from its sinking below 100 *mm*.

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[1] Boyd, Clinical Importance of the Cerebro-spinal Fluid, B. M. J., May 2, 1914.

Heine[1] has found that nystagmus and the so-called neurotrophic corneal affections (herpes, dendritic keratitis, bullous keratitis, dystrophia epithelialis) are symptoms of a meningeal irritation which is often latent and shows itself in increased intracranial pressure. To get a correct expression of the increased intracranial pressure, a careful technique in the lumbar puncture must be followed. Heine believes that the eye symptoms are produced by the increased intracranial pressure, because in a number of cases the reduction of the lumbar pressure exerted a favorable influence on the eye affections. It is possible that this may be an example of not only a latent meningeal irritation, but of a general disturbance of an infectious or toxic character.

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[1] Heine, "Uber die Höhe des Hirndruckes bei einigen Augenkrankheiten," M. m. W., Nov. 4, 1913.

Lues and tuberculosis and post-infectious intoxications (pertussis, influenza) may be of importance in this connection, because iritis is not infrequently added to the corneal affections and to nystagmus an involvement of the optic conducting paths. To answer the question, whether primary eye diseases are able to increase the intracranial pressure, Heine examined a number of corneal affections which differ from the neurotrophic. He found that severe and moderate injuries of the eye in otherwise healthy persons may produce an increased intracranial pressure which persists for a number of months and sometimes causes headache, vertigo and similar cerebral conditions. This would mean that we have not to deal with a meningeal irritation from a simple reflex through the trigeminus, but the cause is a more complicated one and partly of the nature of psychogenic shock, excitement, fear, or worry.

The lumbar pressure in 25 cases was as follows: 150 in 15; 200 in 5; 300 in 5; over 300 in none. In the neurotrophic corneal affections 150 in 30; 200 in 9; 300 in 11; over 300 twice. In nystagmus 150 in 7; 200 in 7; 300 in 9; over 300 twice.

Callosal Puncture.—Increased intracranial pressure may be due to: (1) a large series of developmental disturbances; (2) morbid changes within the cranial cavity, possibly also functional disturbances which act on the vasomotor components. The operation for relief in increased intracranial pressure has for a long time been trephining. Anton and von Bramann have become convinced that trephining in many cases is not successful in accomplishing the desired effect. Moreover, incision or removal of the dura in many cases is made necessary. The brain thus loses an important protective organ and is liable to suffer. An increase of pressure due to increased intraventricular fluid can only be incompletely corrected by trephining. The search for other methods for relieving this pressure, especially in hydrocephalus, has been met by callosal puncture.

For the proper nutrition and the undisturbed function of the brain a free communication must exist between the ventricles and the subdural spaces. In many diseases of the brain this communication is disturbed. A puncture through the roof of the ventricles is easily made.

Hydrocephalus should be operated on early, before the enormous dilatation of the head develops with the associated changes in the brain, which have such serious influence on the intelligence. It seems that the reduction of the intracranial pressure has favorably influenced the mental deterioration. The fluid should be evacuated very slowly and in not too large an amount. The operation has been performed in tumors of the hypophysis, in tumors of the corpora quadrigemina, and of the fourth ventricle, in meningitis and in tower-skull. Von Hippel believes that this operation is generally capable of producing a distinct reduction in pressure. This is shown by an arrest of the choked disk and a reduction of the symptoms of headache, vertigo and vomiting. In the treatment of choked disk von Hippel regards callosal puncture as one of the important methods. It is the least dangerous intervention and is always to be urged when the choked disc can be operated on in the proper, that is early, stage. If the early stage has passed, trephining is still the best operation.

THE DIAGNOSIS OF INTRACRANIAL OPTIC PATH LESIONS[1]

The **chiasm,** owing to its anatomic position and close relation to important structures (p. 11), may be affected in meningitis (hydrocephalus), tumors of the hypophysis, basal or brain tumors, syphilis, fractures, aneurysms and arteriosclerosis.

In basal fractures disturbances of vision are generally not determined, owing to the state of the patient. Arteriosclerotic changes in the blood vessels ascending at the lateral angle of the chiasm may cause pressure on the underlying nerve fibres and produce the unusual condition of binasal hemianopsia. Aneurysms have been known to divide the chiasm and produce a bitemporal hemianopsia.

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[1] Henschen in Lewandowsky, Handbuch d. Neurologie, Vol. III, p. 751.

Acute cerebro-spinal meningitis, meningitis following typhoid fever, pneumonia and other infectious diseases, and acute purulent meningitis disturb vision through optic neuritis as well as by involvement of the chiasm or of the cortex. In many of these cases there are pupillary changes and ocular-muscle paralyses. At autopsy large masses of exudate are found about the chiasm. Paralysis of the ocular muscles and choked disc with iridoplegia suggest a basal origin of the meningitis. Hemianopic disturbances are usually absent in these acute meningitis cases, unless associated with brain abscess. Tuberculous meningitis is often localized about the chiasm. The loss of sight may be explained by the optic neuritis, hydrocephalus, as well as an exudate about the chiasm. Hemianopic defects are absent. Tuberculous tumors have been observed in the chiasm with bitemporal hemianopsia or blindness. Chronic basal meningitis often is accompanied by severe disturbances of sight on account of marked changes in the optic nerve. Serous meningitis and its accompanying internal hydrocephalus may cause a compression of the optic nerves and an optic neuritis from distention of the third ventricle. Cystic distention of the III ventricle has been known to press on the hypophysis and chiasm with the production of symptoms of hypopituitarism.

The firm bony and dural capsule of the hypophysis and the slow growth of these tumors explains why hypophyseal tumors cause but few symptoms. The tumor excavates the sella downward, forward and backward, without producing neighborhood symptoms. In this stage the X-ray alone will make the diagnosis. The tumor then, if it grows upward and enlarges the infundibular opening in the diaphragm, will press on the ventral surface of the chiasm (see pituitary tumors, p. 213). Henschen states that tumors originating from the dura in the neighborhood of the chiasm, have been observed a number of times. They produce visual disturbances, partly by increasing the intracranial volume and by choking the disc, or by pressing directly upon the optic nerve or chiasm. Sometimes they first of all affect the ocular nerves and the surrounding parts. Tumors of the frontal lobe have extended toward the chiasm and caused complete blindness. The diagnosis in these cases is based on the general characteristics of a tumor process, particularly intense localized V nerve headache, and on the visual disturbance. Disturbance of smell and facial anæsthesia are of diagnostic importance. Tumors of the III ventricle may extend to the chiasm and cause blindness.

Basal **syphilis** situated about the chiasm produces symptoms closely resembling hypophysial tumors. The chiasm is the site of predilection for a syphilitic process. Lues appears in this position in the form of a diffuse chronic basilar meningitis, often accompanied by pronounced vascular changes and gummata. It may extend over the entire base of the brain to the medulla and involve the basal nerves. It offers many varying clinical forms and the chiasmal symptoms are often associated with changes in the eyes (choroiditis, retinitis) and in the optic nerve (neuritis). Though basal syphilis is apt to be situated about the chiasm, it does not affect the chiasm itself as frequently as it does the **basal ocular nerves.** Many years before its development, there may be transitory paralysis of the ocular nerves, in the order of frequency—III, VI and IV, with simultaneous characteristic headache. Among the functional eye changes, as Uhthoff has shown, hemianopsia occupies the first place, the bitemporal form being more frequent than the homonymous. The central localization of the process explains the absence of ophthalmoscopic changes.

The changes in the field vary and are often complex, especially when an optic nerve or tract is affected in addition to the chiasm; there may be a relative scotoma, monocular temporal hemianopsia, bitemporal hemianopsia, temporal hemianopsia with blindness in one eye, blindness in one eye with nasal hemianopsia of the other, blindness in both eyes which does not but exceptionally remain permanent.

Chiasmal syphilis shows frequent variations in vision, so that the blindness is often only transient, especially if proper treatment is energetically carried out. In this respect it differs from the visual disturbance in tabes and paresis, where the loss in vision usually continues steadily. Simple gray degeneration of the optic nerves, without inflammatory symptoms, is unusual. An important associated symptom is paralyses of the basal nerves, and particularly of the ocular nerves. Lesions of the III nerve, bilateral or monolateral, and of the VI, usually bilateral, rarely of the IV, usually one-sided, are frequent. Of the general symptoms, headache must be mentioned. It is very severe, and may be accompanied with vomiting or periods of excitement, insomnia and stupor. Diabetes is often present.

Optic Tract.—The main symptoms of a tract lesion are homonymous hemianopsia and hemiopic pupillary reaction. The causes are hemorrhage, softening, tumor or lues. Owing to its small size, the tract is rarely the starting point of a pathological process, excepting the luetic ones, which probably arise from the vessels and the meninges, the tracts are usually involved by extension or by "distant" action. Hemianopic defects are unusual in meningitis. The tract is rarely involved as a result of hemorrhage or thrombosis. Syphilis of the tract is not infrequent, and occurs about twice as frequently as at the chiasm. Its most frequent site is directly back of the chiasm and may extend to the latter. It may even be bilateral, in which case it will affect the chiasm as it passes from one tract to the other. Occasionally the syphilitic process affects the whole length of the tract and, just as in the chiasm, the visual field defect varies. A distinct hemiopic pupillary reaction is rarely seen, probably because all the fibres in the tract are not equally affected. If the process has existed for any length of time, the disc shows an atrophic condition without preceding papillitis. The ocular nerves are often affected as well as the V nerve. If the process extends to the peduncle, it will cause a paralysis of the opposite half of the body. The general brain symptoms are the same as in the chiasm. The hemianopsia may sometimes be restricted to a quadrant, as has been observed in three cases of tumors. The hemiopic pupillary reaction is of importance for diagnosis when it is positive rather than negative.

Central Ganglia.—As there are no optic fibres in the pulvinar nor in the anterior corpora quadrigemina, a lesion of these two structures does not produce an alteration in the visual field, though possibly of the pupil. All disturbances of the field from lesions in this region must be due to a disturbance of the function of the external geniculate body. Owing to its small size, pathological processes are rarely limited to the external geniculate ganglion but invade the neighboring structures. These are: (1) the auditory tract passing from the posterior quadrigeminal body through the internal geniculate body to the temporal lobe; (2) the sensory tract passing through the posterior segment of the internal capsule from the lemniscus to the posterior central convolutions; (3) at a greater distance away are the nuclei of the ocular nerves; and (4) the middle division of the internal capsule which contains the motor tract.

Hemorrhages are frequent in the adjoining internal capsule or pulvinar. This explains the hemiplegias which are accompanied by hemianopsias. The hemorrhage in these cases often compresses the geniculate body so completely that complete hemianopsia results. Henschen states that owing to its free position, the function is frequently restored, though the dorsal portion may present a permanent atrophy which shows itself in the form of an inferior quadrant hemianopsia. Tumors in the central ganglia may produce hemianopsia from pressure on the geniculate body or on the tract. The entire thalamus can be destroyed without causing a visual disturbance. It is of course difficult from the accompanying eye changes to decide whether the lesion is situated in the tract or in the geniculate body, though Henschen believes that the lesion of the geniculate body shows itself by homonymous hemianopsia, subsequent optic atrophy, absence of hemiopic iridoplegia and, according to the extent of the lesion, hemianæsthesia (or motor hemiplegia) and deafness on the opposite side. In a tract lesion hemiopic iridoplegia is present.

Affections of the Optic Radiation and Visual Cortex.—As has been explained in the anatomical part, a lesion only in the lateral part of the occipital medulla, the stratum sagittale occipitale laterale (inferior longitudinal fasciculus) can cause visual disturbances in the form of homonymous hemianopsia, complete, quadrant, or as scotomata, without hemiopic pupillary reaction.

Visual disturbances may occur as a result of an injury to the parietal region. As the most dorsal fibres of the visual path attain the height of the lower portion of the angular gyrus, a lesion in this region can cause either a quadrant or a complete hemianopsia and occasionally no visual disturbance. The lesions which have caused these symptoms are: shot wounds, hemorrhages, softenings, abscesses, and particularly tumors. In the last condition, during the development of the tumor, the visual disturbance may begin with a quadrant defect in the lower visual field and then forms a complete hemianopsia. A number of these cases have been observed by Henschen.

The most important lesion in the temporal lobe is a temporal abscess of otitic origin. This easily involves the optic paths and produces homonymous hemianopsia with disturbances of speech if it be on the left side; furthermore, through distant action, disturbances of motility (facial nerve, hemiplegia) and III nerve paralysis (ptosis, dilated pupil, oculomotor paralysis) not infrequently choked disc and general symptoms. This hemianopsia is not accompanied by hemiopic pupil. Its presence in otitic suppurations is of the greatest diagnostic importance, because the hemianopsia is a definite sign that the abscess is situated within the brain substance, while in the superficial otitic affections of the meninges hemianopsia has not been observed. Hemianopsia has been observed in tumors in the temporal lobe, where it is an indirect and uncommon symptom. Softenings which affect the optic path in the temporal lobe are frequent. They result from a lesion of the posterior cerebral arteries.

If from the presence of homonymous hemianopsia a process in the occipital neuron of the optic tract is suspected, it may be possible from the accompanying symptoms to determine whether the medulla of the parieto-temporal lobe or of the occipital lobe is affected. This is difficult if the lesion is in the right hemisphere, as the right parietal and temporal lobes do not possess characteristic functions. A lesion of the internal capsule would show itself by hemiplegia with hemianæsthesia. Central deafness or an otitic process speaks for the temporal lobe. If the process is on the left side, an important symptom is aphasia. Henschen draws particular attention to two further features. Processes in the parietal lobe are not infrequently characterized by a quadrant hemianopsia downward, while softenings in the occipito-temporal lobe are apt to be accompanied by quadrant hemianopsia upward. A careful study of the visual disturbances, their development and course, together with the accompanying symptoms, frequently make it possible to make an exact diagnosis. Tumors occur in the parietal lobe, and abscesses, usually otitic in origin, are situated in the temporal lobe, laterally from the optic paths; hemorrhages occur in any position of the optic paths.

The fibres of the visual path in the **occipital medulla** are no longer crowded together, and lesions in this region consequently affect a smaller number of visual fibres, resulting in circumscribed defects in the field in the form of quadrants or sectors or small scotomata, which are always hemianopic.

Disturbance of circulation in the occipital lobe may affect vision. **Scintillating scotoma** belongs to a group of functional disturbances which some authors believe to be due to a disturbance of circulation, an angioparalytic condition of the vessels or an angiospasm. This condition is characterized by migraine-like attacks accompanied with flashes, by homonymous defects in the visual fields, sometimes for color, in others quadrant in outline or occupying the entire half of the field; the macular region is usually preserved. The hemianopsia is generally transitory; occasionally it persists. The seat of this lesion is generally placed in the occipital lobe, and this is also favored by the presence of hallucinations with the hemianopsia. Sudden blindness occurring in uremia has its seat in the occipital lobe and probably results from disturbances of circulation.

Owing to its exposed position, the occipital lobe is frequently injured by **trauma.** Henschen states that in cerebro-spinal meningitis amaurosis with preserved and prompt pupillary reaction is not very unusual. Abscesses occur in the occipital lobe, particularly after trauma, and are followed by visual disturbances. The abscesses may also be otitic, metastatic or traumatic. The resulting visual disturbances are homonymous hemianopsia, bilateral hemianopsia, inferior hemianopsia and macular defects.

One of the results of the trauma, usually after fracture or shot wound, is a hemorrhage in the occipital lobe which causes defects in the visual field. The patient, after the accident, is usually unconscious and on regaining consciousness he is found completely blind for a varying length of time, followed by a period of soul blindness; then the vision gradually returns, the field clears up and a partial scotoma remains.

Tumors are either metastatic or primary, the latter usually extending from the parietal lobe. Henschen states that they frequently press from above downward, affecting the dorsal visual fibres, and produce a hemiopic defect, especially of the ventral quadrant. Through pressure and the associated choked disc a concentric contraction of the visual field is also present. As the tumor grows, the defect increases until it completely occupies half of the field. Tumors which start in the cortex cause at the beginning symptoms of irritation in the shape of color hallucinations or homonymous light and figure hallucinations which are distinctly localized in a certain direction or in a definite quadrant.

Vascular changes in the occipital lobe may result in thrombosis and consecutive softening. A variety of visual disturbances may result.

I. Softenings in one occipital lobe may cause no change in vision, particularly when the lateral part is affected. If the tract or the visual cortex is involved, hemianopic changes result of the following varieties; homonymous complete hemianopsia; quadrant hemianopsia; or a number of small scotomata.

II. Bilateral softenings, where both occipital lobes are affected: Two mesial cortical surfaces are usually involved from a thrombosis of both posterior cerebral arteries, and bilateral homonymous hemianopsia, varying in size and shape, result. Henschen describes the following clinical forms: (*a*) With complete persistent blindness, where the entire visual cortex has been completely destroyed. About 25 cases of this variety are known. (*b*) With transitory blindness of the macular field, which later clears up while the peripheric field remains blind (12 cases). (*c*) Bilateral homonymous hemianopsia with a persistent fixation field (23 cases). In these cases the macular field is preserved in its entire or in its partial extent. (*d*) Other forms of visual disturbances, particularly color blindness.

In these cases of bilateral blindness the patients are not conscious of their blindness and their optical memory is preserved. In many cases visual hallucinations were present. If a part of the visual field remains, the patient is unconscious of it, and has also lost the sense of orientation and of color.

II. DISEASES OF THE NERVOUS SYSTEM

MENINGES

Pachymeningitis Interna Hemorrhagica—Dural Hematoma.—This condition occurs generally in adults suffering from progressive paralysis, arteriosclerosis, etc., or, exceptionally, after birth-traumas. The symptoms depend on the size and the location of the hematoma. The entrance of blood into the optic nerve sheath causes optic neuritis (choked disc) which is the ocular characteristic of this condition. As in the majority of cases this is one-sided, it is of diagnostic value in cases of brain tumor or hemorrhage following injuries to the skull where the optic nerve changes usually are bilateral. Retinal hemorrhages have been observed particularly in small children. Ocular-muscle paralyses are not a part of this condition unless there are complications. Conjugate deviation of the eyes, however, is a frequent symptom combined with rotation of the head to the same side and with nystagmoid movements when looking in the opposite direction. The pupils show no particular change.

Sinus Thrombosis.—Thrombosis of the cerebral sinuses occurs broadly speaking in two forms. The first, primary or *marasmic*, is very unusual, occurring at the two extremes of life in patients whose circulation is weakened after an exhausting illness, diarrhœa, pneumonia, etc. The longitudinal sinus is the one usually affected though in the severe and fatal cases other sinuses are also found involved. The diagnosis of this form of sinus thrombosis is difficult and the condition does not cause noticeable eye symptoms.

More important is the second variety, the **infective** or inflammatory form. This is generally observed in adults after fractures of the skull or infected scalp wounds, after middle ear disease where the sigmoid sinus is involved or after conditions which lead to cavernous sinus thrombosis.

Sigmoid sinus thrombosis is the most frequent and constitutes one of the serious intracranial complications of purulent otitis and in its diagnosis disturbances of vision and changes in the eye grounds are of great importance.

In the interpretation of the eye changes it must not be forgotten that septic thrombosis is complicated in many cases with meningitis or brain abscess.

Uhthoff found changes in the eye grounds in more than one-half of the cases. These consisted in choked disc, optic neuritis and hyperemia. Optic atrophy is a very unusual symptom and does not belong to this clinical picture. In most of these cases of sinus thrombosis where chocked disc or optic neuritis are present, the sinus thrombosis is complicated with either meningitis or brain abscess. Uncomplicated otitic sinus thrombosis gives rise to optic neuritis in only a small proportion of cases. On the other hand, optic neuritis and choked disc have been occasionally observed in uncomplicated mastoiditis and even in uncomplicated purulent otitis; as a general rule these ophthalmoscopic changes nearly always indicate a severe intracranial complication. It is probable that these ophthalmoscopic changes are due to an associated serous meningitis (hydrocephalus). The ophthalmoscopic changes are usually bilateral; sometimes they appear earlier and more intensely on the side of the ear affection. It is also to be noted that after successful operation the ophthalmoscopic condition may not retrograde but very slowly and in some cases the condition in the eye grounds has become worse without in any way affecting the prognosis.

According to Uhthoff, the ocular muscles were found affected in about 12 per cent. of the cases. In more than one-half of these cases the cavernous sinus was involved. As has been shown, these nerves are directly affected while in the wall of the cavernous sinus. This explains how frequently the abducent nerve is involved, practically in two-thirds of all the cases. Other intracranial complications must explain the ocular paralysis in the cases where the cavernous sinus was not involved. Extension of the disease to the apex of the petrous pyramid can produce a direct lesion of the ocular nerves, particularly of the VI nerve. The III nerve was involved next in frequency to the VI. The involvement was only partial and the elevator of the upper lid was the branch most frequently affected. Isolated IV nerve paralysis and conjugate deviation of the eyes were not observed. Nystagmus was not an infrequent symptom, though it is probably due to a complicating affection of the labyrinth.

The infective thrombosis may extend through the petrosal sinus and involves the cavernous sinus where œdema of the eye lids, slight exophthalmos and other symptoms characteristic of cavernous sinus thrombosis appear.

Thrombosis of the **cavernous sinus** also occurs primarily and this is the form of greater importance to ophthalmologists. Herman Knapp[1] was the first to clearly recognize thrombosis of the cavernous sinus in an article published in 1868. Primary cavernous sinus thrombosis usually results from an inflammation in the orbit extending along the ophthalmic veins which empty directly into the cavernous sinus. According to Macewen,[2] other causes are erysipelas of the face with secondary phlebitis of the facial and ophthalmic veins, an infective inflammation in the mouth, nose or throat, or an ulceration of the tonsils and of the pharynx, which produces a thrombosis of the veins of the pterygoid plexus. Caries of the body of the sphenoid causing infective meningitis has also led to thrombosis of the cavernous sinus. There are a number of infective processes of the nasal cavities and accessory sinuses which have been known to produce a thrombosis of the cavernous sinus. Lesions of the pharynx and the tonsils have also been followed by this thrombosis. A

[1] Knapp, Arch. f. Ophthalmologie, 1868, Vol. XIV, p. 220.

[2] Macewen, Diseases of the Brain and Spinal Cord, p. 226.

further cause is alveolar periostitis after lesions of the teeth. One cavernous sinus is usually first affected, then in more than half of the cases the process extends through the circular sinus to the cavernous sinus of the other side. This explains how the symptoms become bilateral and that the process in one side may be more marked than on the other side. Macewen suggests that this alternation of the symptoms is a diagnostic distinction between an inflammation confined to the orbit and a cavernous sinus thrombosis.

Pain is one of the early symptoms. Symptoms depend upon venous obstruction and on paralysis due to pressure on the nerves situated in the cavernous sinus. The first produces exophthalmos, œdema of the eyelids, and chemosis. The second is responsible for ptosis, strabismus and pupillary manifestations. The exophthalmos may vary to a marked degree. It is particularly marked when there is an orbital phlebitis and retro-orbital abscess. The ophthalmoscopic symptoms are of interest, though they are only present in 50 per cent. of the cases. This is probably due to the collateral circulation of the orbital veins. If the cavernous sinus alone is involved and the process does not extend to the orbital veins and to the orbit, changes in the eyegrounds may be absent and exophthalmos will then also be absent. If there is pronounced optic neuritis an involvement of the orbit may be assumed (thrombosis of the orbital veins, abscess of the orbit, protrusion of the eyeball and œdema of the lids). On the other hand, it is interesting to note that the so-called apoplectic retinitis as we find after a thrombosis of the retinal vein, has practically never been observed. A metastatic purulent ophthalmia is also very unusual and if it is present it is probably the evidence of a metastatic infection from some other part of the body. In severe infections of the orbit from a phlebitis of the veins, purulent inflammation in the interior of the eye does not occur. The sight of the eye is lost on account of the severe damage which is done to the optic nerve in the orbit. The pronounced paralysis of the ocular muscles indicates the direct invasion of the nerves in the sinus wall. This also explains the involvement of the first branch of the V nerve. The III nerve is the most frequently implicated and ptosis is a nearly constant symptom, though it is sometimes difficult to decide whether the ptosis is not simply due to the œdema. In the advanced cases ophthalmoplegia is complete. Vision is affected and amaurosis from pressure on the optic nerve appears late.

Exophthalmos is the most frequent eye symptom. This was found in 72 per cent. of the cases. If this is particularly marked with distinct loss of motility on the part of the eyeball, an extension of the thrombotic process to the ophthalmic veins can be assumed. The symptoms gradually develop and become pronounced in one eye, then the other eye becomes affected with exactly the same symptoms. With the involvement of the second eye the symptoms of the first eye often subside. The implication of the second eye is a sure indication of a thrombotic extension to the opposite sinus. In practically 50 per cent. of the cases, if the cavernous sinus on one side is involved, the other side will also be involved. The time at which the exophthalmos and cerebral symptoms appear are of importance in estimating the nature of the process. Thus, when the orbital symptoms are primary the involvement of the cavernous sinus is secondary, while if an exophthalmos with swelling of the lid follows the onset of cerebral symptoms, the reverse order is present. These orbital symptoms do not occur in other purulent intracranial processes and are not found in purulent meningitis, so that an inflammatory exophthalmos is a very important symptom of a thrombosis of the cavernous sinus, especially if it is associated with a dilatation of the frontal veins, optic neuritis and paralysis of the ocular muscles. The pupils do not offer any particular diagnostic change. In addition to these local symptoms, general symptoms of sinus thrombosis—chills, high fever, vomiting, and secondary complications—are present. The great majority of the cases end fatally.

Tuberculous Meningitis.—The pathological lesion in tuberculous meningitis is never purulent but generally a fibrino-plastic exudation with the presence of many miliary tubercles. These changes are marked at the base of the brain, thus affecting the large cranial nerves, particularly the optic paths and the motor ocular nerves, and explains the frequency of the ocular symptoms. After a prodromal period characterized by irritability, prostration, headache, apathy and vomiting, the disease begins with fever and severe cerebral symptoms (headache, vomiting, rigidity of neck, delirium, convulsions and stupor). The outcome is usually death after a few weeks. There are rapid cases and in some cases the disease assumes a chronic course, lasting for months or for years; in some cases the tuberculous meningitis seems to get well.

Ophthalmoscopic changes in the eye grounds occur in nearly one-half of the cases. The most important one of these is **optic neuritis** which is present in half of the cases. The optic neuritis shows a clouding of the disc, a blurring of the margins and increased congestion without distinct prominence. It is stated that the frequency of optic neuritis in tuberculous meningitis is explained by the more or less chronic course of this disease. At the same time, opportunities for observing a neuritic optic atrophy in tuberculous meningitis are unusual. The condition does not generally lead to blindness. The typical choked disc is an unusual symptom; it occurred in 5 per cent. of Uhthoff's cases and was always associated with the presence of a solitary tubercle in the brain.

Uhthoff speaks of marked loss of vision in tuberculous meningitis with relatively slight ophthalmoscopic changes in the optic nerves. This has been observed in 9 per cent. of the cases and is regarded as a descending optic neuritis where the lesion of the optic paths is so far back as not to cause changes in the nerve head. Caution must be exercised in placing great diagnostic importance to changes in the retinal veins and a reddening of the discs when other pathological conditions (œdema and clouding) are absent in the optic nerve and retina. Retinal hemorrhages are unusual in this condition, though they have been observed by a number of authors, and were generally present in conjunction with optic neuritis.

Choroidal tubercles in tuberculous meningitis have been observed in a comparatively small percentage of cases. Conheim was the first to draw attention to the striking frequency of choroidal tubercles in general miliary tuberculosis. These small grayish-yellow, somewhat prominent, sharply-defined, areas in the choroid are principally situated in the posterior pole of the eye and present a characteristic ophthalmoscopic picture. In Uhthoff's statistics tubercles of the choroid were found in 10 per cent. of the cases of tuberculous meningitis, and in one-half of these the condition was complicated with optic neuritis. Other authors have found the percentage to be somewhat larger, 19 per cent., though in many of these cases a general miliary tuberculosis was found at autopsy. Jessup reports in 15 cases of choroidal tuberculosis tuberculous meningitis in 14, and Carpenter and Stevenson also found choroidal tubercles to be frequently present, and in 36 cases there was an associated tuberculous meningitis in 26. Optic neuritis occurs much more frequently in tuberculous meningitis without choroidal tubercles than with them. This proves that these two conditions are not interdependent, and shows that the choroidal tubercles do not originate from an optic neuritis or a retrobulbar tuberculous perineuritis where infection travels directly from the brain to the eye along the optic-nerve sheaths. Choroidal tubercles even when they are complicated with optic neuritis in tuberculous meningitis are the result of a metastatic hematogenous infection, and the presence of choroidal tubercles in tuberculous meningitis points to a complicating general miliary tuberculosis. The presence of choroidal tubercles in general miliary tuberculosis is therefore much greater than in tuberculous meningitis. The statistics have shown a percentage of 42 per cent.

Uhthoff found a **disturbance of the ocular muscles** in 50 per cent. of the cases. The III nerve seems to be most frequently involved, though it is not so frequently affected as in syphilitic basal meningitis. Bilateral III nerve paralysis has practically never been observed. The lesion usually found is a paresis of various branches in 12 per cent. and an isolated ptosis in 16 per cent. The pathological process in tuberculous meningitis, according to Uhthoff, does not affect the nerve trunks themselves, but rather to disturb the function from the presence of an exudate about the nerve trunk, thus producing slight paresis and particularly a moderate ptosis. The abducent nerve is affected in about 15 per cent.; a bilateral involvement is extremely unusual. A IV nerve paralysis and ophthalmoplegia apparently do not belong to this clinical picture. Conjugate deviation of the eyes with occasional rotation of the head to the same side is an infrequent symptom. Nystagmus is also an infrequent symptom.

The pupils show changes of importance in tuberculous meningitis. A disturbance of light reaction has been found in 15 per cent. of the cases and was usually seen in patients in deep stupor. A difference in the size of the pupils, independent of the illumination, has been described by a number of authors. During Cheyne-Stokes respiration dilatation of the pupils has been observed at the onset of the respiratory phenomenon and a contraction during the period of rest.

Epidemic Cerebro-Spinal Meningitis.—This condition, as its name implies, occurs in epidemics, though sporadic cases appear from time to time. It is a disease of youth, occurring most frequently in children under 10 years of age. The mortality varies with the epidemic from 33 to 80 per cent. The infectious organism is the meningococcus and it is regarded as a direct extension from the nasopharynx. The period of incubation is short, the course of the disease is either an unusually rapid one or the ordinary acute form. There are apparently many variations from the typical course. A number of epidemics have been carefully studied in regard to the frequency of their eye complications.

Conjunctivitis has been observed and is probably an endogenous infection. The cornea has been involved in a very small number of cases and its lesion is not uniform. Dendritic keratitis has been described and it probably results from conditions similar to febrile herpes which occurs so frequently in cerebro-spinal meningitis. Secondary infection in dendritic keratitis can, of course, lead to very severe ulcerating processes in the cornea, though a particularly severe ulcerating form is the one which is spoken of as keratitis e lagophthalmo or keratitis xerotica. This has been described a number of times and always starts in the lower third of the cornea, that part which corresponds to the palpebral opening.

Optic neuritis is not a frequent symptom, and was present in about 17 per cent. of the cases. The typical choked disc is very unusual. The pathological examination of cases of optic neuritis in cerebro-spinal meningitis have shown in some the signs of a descending neuritis, indicating the extension of an inflammation from the brain along the nerve sheath, while in others changes of this character were absent. In a case which Uhthoff examined the symptoms of a descending peri-neuritis were not marked and the changes seem to be due rather to an associated internal hydrocephalus with bulging of the floor of the third ventricle. Uhthoff is therefore inclined to regard the method of development of an optic neuritis in cerebro-spinal meningitis as not settled, as the condition may be due to either a true descending neuritis or to some other condition, like increased intracranial pressure. Disturbances of vision without ophthalmoscopic changes are not infrequent and are usually the result of the presence of exudative processes affecting the optic pathways or the visual cortex in the occipital lobe.

The symptom which is of particular interest to us is **metastatic ophthalmia.** This was found present in about 4 to 5 per cent. of the cases. The clinical picture has certain characteristic traits. The external inflammatory symptoms are slight; there is a yellowish reflex from the depth of the eye with moderate iritis. Even if the inflammatory symptoms are severe, they may improve and the shape of the eye ball is preserved, though it becomes somewhat smaller with marked flattening of the anterior chamber and bulging of the iris, a reduction of the intraocular tension and the absence of marked pain. The condition is furthermore one-sided. Other infectious organisms may produce similar changes, though as a rule these organisms, if they lead to metastatic ophthalmia, produce a clinical picture with very much more marked symptoms, frequently resulting in panophthalmitis with perforation of the eye ball and phthisis. In cerebro-spinal meningitis the metastatic inflammation of the eye may run a comparatively benign course and the sight of the eye need not be entirely destroyed. There are cases of comparatively mild iritis and iridochoroiditis, though this is a very unusual occurrence. Though metastatic ophthalmia in cerebro-spinal meningitis is monolateral in most cases, both eyes are affected in a small percentage and sometimes, to an unequal degree. Metastatic ophthalmia may be one of the first symptoms of the general infection and it also occurs in the late period. It does not necessarily mean an intense general infection, as it is seen in cases where the general symptoms are unusually mild. The infection is hematogenous, affecting either the retina or the choroid; the intraocular inflammation is very rarely due to a direct inflammatory extension along the optic nerves.

Disturbances of the ocular muscles are important and occur in 15 per cent. of the cases. The VI nerve is most frequently affected, presumably on account of its long course at the base of the brain. The III nerve is affected in the form of a partial paralysis, particularly as a ptosis of moderate degree; the IV nerve seems to escape involvement in this disease. Complete paralysis of all the muscles has been observed. Conjugate deviation of the eyeballs is somewhat more frequent. Nystagmus is not an important symptom. Pupillary changes are neither frequent nor characteristic.

Otogenic Purulent Meningitis.—Acute purulent meningitis has the tendency to spread rapidly and diffusely. It may start at the base or at the convexity of the brain and rapidly involve the entire surface. When the meninges are inflamed the underlying brain tissue is often involved in the form of a meningo-encephalitis. A large proportion of the cases of purulent meningitis are due to infection from the ear and from the petrous pyramid. The infection in these cases either extends through the roof of the middle ear or antrum to the middle cranial fossa or through the agency of the sigmoid sinus and the internal auditory meatus to the posterior cranial fossa. Uncomplicated otitic meningitis is infrequent in small children and generally appears in the second and third decade. Its course is usually extremely rapid or it may be more prolonged and insidious, in which case it will last for a number of weeks before death results. The symptoms depend upon whether the process is localized to the base of the brain in the middle and anterior fossa or in the posterior cranial cavity. In the former the eye symptoms are, of course, the more frequent.

As for the frequency of otitic meningitis compared to other forms of meningitis, Uhthoff quotes Pitt, who found in 9000 autopsies 25 otitic meningitis, 162 tuberculous meningitis, and 133 of other causes, including lues. In 78 cases that came to autopsy at the Munich Child Hospital, 57 were tuberculous, 15 were epidemic cerebro-spinal meningitis, and only 1 was otitic.

Otitic meningitis is less frequent than the other otitic cerebral complications (abscess of the brain, sinus thrombosis) and in many cases these conditions are combined.

Changes in the eye grounds, particularly **optic neuritis** in uncomplicated otitic meningitis, is very unusual. This is probably due to the shortness of the course of the disease and no opportunity is given for the development of changes in the optic nerve heads. True choked disc is even more unusual in otitic meningitis than optic neuritis. If it be present, it always points to an intracranial complication. The onset of optic neuritis following aural suppuration indicates an intracranial complication and even if it is only slightly developed furnishes a definite ground for operation. The extradural abscess which sometimes is seen in ear diseases may cause an optic neuritis combined with general brain symptoms, headache, increased intracranial pressure, stupor, vomiting, retarded pulse, even when a definite intracranial complication is not present. It may be stated that changes in the disc are present in about 50 per cent. and are of value in the diagnosis of otitic intracranial complications. At the same time they do not furnish any definite conclusion on the nature or on the site of the complication. It does not always follow if the changes are more marked on one side or are monolateral that that indicates the side affected, though in many cases this conclusion is justified. After an operation on the skull in ear disease the ophthalmoscopic changes may increase for a time, though the patient be on the road to recovery.

The **facial nerve** is probably affected in otitic meningitis more frequently than any other of the basal cranial nerves. Of the ocular nerves, the **abducent nerve** is most frequently involved. This may be partly due to its location at the apex of the petrous pyramid where it seems particularly vulnerable. Oculomotor involvement, generally only partial, is very unusual and not nearly as frequently observed as in other forms of meningitis, particularly the more chronic forms such as the syphilitic variety.

Basal paralyses of the motor ocular nerves are more frequently present in uncomplicated otitic meningitis than optic neuritis, though monolateral or bilateral ophthalmoplegia have not been observed. The involvement of the ocular nerves is of some value in diagnosis; thus in the uncomplicated otitic meningitis the abducent nerve is the most frequently affected and the paralysis is incomplete. In brain abscess in the temporal lobe the III nerve of the same side is sometimes involved.

The changes in the pupils are of no particular moment. Metastatic ophthalmia is a very unusual complication, differing in this respect from epidemic cerebro-spinal meningitis. The development of this intraocular inflammation always indicates a general infection of the body, as is not due to an extension of an infectious meningeal process from the brain. In fact if an ophthalmia occurs during a meningitis it rather indicates that the purulent meningitis is not a local process but is due to a general infection. Even in the cases where purulent meningitis with septic sinus thrombosis leads to an inflammatory process in the orbit (orbital abscess and exophthalmus) a purulent inflammation in the interior of the eye practically never results.

It is sometimes very difficult to distinguish between otitic meningitis and tuberculous meningitis because a tuberculous osteitis of the petrous pyramid may be the direct starting point of a tuberculous meningitis, and a tuberculous otitis may be associated with a tuberculous meningitis as an independent lesion. The presence of choroidal tubercles would be positive proof, though it is very unusual. Inflammatory changes of the discs occur more frequently in the tuberculous than in the simple otitic meningitis. On the other hand, otitic meningitis is complicated with sinus phlebitis and brain abscess in 50 per cent. of the cases, which would lead to ophthalmoscopic changes.

Meningitis which occurs after certain **infectious diseases,** such as typhoid fever, pneumonia, influenza, scarlet fever, measles, sometimes produce eye symptoms. Probably the most frequent is the meningitis in typhoid fever. It does not differ from other purulent forms of meningitis. Optic neuritis has been noted in a number of cases. It must be remembered that the optic neuritis may be present without meningeal symptoms, though if in the course of a typhoid fever visual disturbances and changes in the optic nerve develop, an intracranial complication, particularly meningitis, is present. Choked disc has not been observed; paralyses of the ocular muscles are also unusual and are due to an intracranial complication (meningitis), though a direct peripheric disease of the ocular nerves through the typhoid toxin is possible.

In pneumonia the clinical course is frequently complicated with pronounced cerebral symptoms, and here again, if ocular symptoms, optic neuritis, paralyses of the ocular muscles, occur under these conditions, meningeal changes are probably present. In the case of influenza, optic neuritis has been observed without the presence of an intermediary meningitis.

Chronic Meningitis Simplex.—There is unquestionably a chronic form of meningitis which is not purulent and which frequently terminates in recovery. It is of importance in explaining certain ocular changes. Ocular symptoms may be due to exudative meningeal changes or to serous meningitis (internal hydrocephalus) which so frequently complicates a plastic meningitis. Investigations in recent years have particularly emphasized the importance of this last cause. Chronic meningitis, if it affects the convexity of the brain, produces but few ocular symptoms. If the base of the brain is affected the aqueduct of Sylvius is not infrequently obstructed and a secondary ventricular hydrops results. Chronic meningitis occurs particularly in children. The most frequent ocular changes are visual disturbances; in fact visual disturbance with cerebral symptoms constitute the picture of chronic meningitis as v. Graefe was the first to point out in 1860. In most of the cases, in addition to the loss of sight, there are changes in the eye grounds. Optic neuritis has been observed, in other cases atrophy of the optic nerves was noted at some later time, the result of a descending degeneration.

Wunderli[1] and Leber believe that an optic neuritis may sometimes be caused by a latent chronic meningitis even when the cerebral symptoms are slight and transient. Ophthalmologists frequently see cases with optic atrophy and marked visual disturbances where the loss of sight began years previously with cerebral symptoms and the patients have since recovered, with the exception of the visual disturbance; these are regarded as evidences of a chronic meningitis. Hemiopic defects of sight have not been observed in this condition. Chronic meningitis may exceptionally produce a central cortical loss of sight with preserved pupillary reaction.

The prognosis of sudden and marked loss of sight in meningitis is not necessarily bad, particularly if the ophthalmoscopic condition is normal and the pupillary reaction is preserved, as sight may still be restored after months. Transient loss of vision in meningitis may be due in part to the internal hydrocephalus.

The ocular muscles are less affected in chronic meningitis than in other forms of meningitis. The VI nerve is the most frequently involved and the III nerve comes next. There are practically no other ocular symptoms of importance.

Serous Meningitis and Hydrocephalus.—An abnormal increase of serous fluid either within the ventricles of the brain or on the outside of the brain between the surface and the dura is accompanied by increase of intracranial pressure. The latter localization is very much less frequent than the former, though both are often combined. Hydrocephalus is not necessarily a serous meningitis, but may be simply a transudate from a mechanical obstruction to the large veins or the openings leading from the third to the other ventricles. These obstructions are known to result from basal meningitis and are usually the cause for the acquired hydrocephalus in small children. Hydrocephalus is also caused by general diseases, like nephritis, cardiac insufficiencies, anemias, rickets, etc. The distention of the ventricles, internal hydrocephalus, is divided into obstructive and non-obstructive.

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[1] Quoted from Uhthoff, p. 843.

The obstructive hydrocephalus is caused by a closure of the foramina of Monro and of Magendie which in most cases is due to a chronic meningitis.

The non-obstructive can be divided into (1) where there is over-secretion from the choroid plexus and (2) insufficient absorption in the subarachnoid space. The recent investigations of Dandy and Blackfan[1] have shown that with the injection of phenolphthalein it is possible to distinguish between these causes. The form which is most amenable to operative treatment is the obstructive, and in this Elsberg recommends callosal puncture.

The presence of the abnormal quantity of fluid in hydrocephalus may exert a deleterious action upon the visual function. If this process develops acutely with symptoms of intracranial pressure, with fever, etc., serous meningitis is diagnosticated. The cerebro-spinal fluid is under pressure and contains albumen. Increased cerebro-spinal fluid under pressure and containing albumen is found in chlorosis, uremia, sinus thrombosis, etc. Quincke believes that it may accompany migraine. Uhthoff found that **changes** in the **optic-nerve heads** were frequently present, consisting in papillœdema, optic neuritis and simple optic atrophy; the choked disc was slightly the most frequent. It is well known that in brain tumor with choked disc an internal hydrocephalus is a constant factor. The age of the patients with choked disc in hydrocephalus was usually over fourteen. The elasticity of the skull in very young children prevents the development of a true choked disc as increased intracranial pressure separates the cranial bones. This is particularly noticeable in the congenital hydrocephalus. In cases of hydrocephalus where there has been a spontaneous discharge of cerebro-spinal fluid from the nose a papillœdema has not been observed. Choked disc occurs more frequently in the acute form of hydrocephalus, or serous meningitis, than in the chronic form.

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[1] American Journal for Diseases of Children, 1914, p. 406.

Optic neuritis and neuritic optic atrophy were observed second in frequency. This ophthalmoscopic change has not been observed in children with congenital hydrocephalus under one year, though optic neuritis is more frequently present in chronic hydrocephalus than choked disc. Optic atrophy without any signs of preceding inflammation was observed in about the same proportion of cases. These were all cases of the chronic form of hydrocephalus and most frequently in children under ten years of age, nearly one-half of the cases were children under the age of one. It seems likely that the optic atrophy is the result of a pressure atrophy through a distention of the floor of the third ventricle. Uhthoff believes that this was definitely shown in at least two-thirds of the reported cases.

Internal hydrocephalus is that form of intracranial disease which most frequently leads to the ophthalmoscopic picture of atrophy of the optic nerves without preceding neuritic symptoms. Visual disturbances without ophthalmoscopic changes are relatively frequent in internal hydrocephalus. The disturbances of the ocular muscles are of less significance in this condition than changes in the optic nerves. At the same time the VI nerve is the most frequently involved and in one-third of the cases changes in the optic nerve (choked disc and optic neuritis) were present. The oculomotor nerve is less frequently involved. Paralysis of associated movement and nystagmus are not infrequent in internal hydrocephalus. That nystagmus is sometimes a pressure symptom is shown by its disappearance after lumbar puncture as numerous observations have shown.

Changes in the pupil are of little diagnostic importance. The only other remaining eye condition is a protrusion of the eyes, an exophthalmos which is due to a downward displacement of the upper orbital wall.

BRAIN

Hemorrhage.—The site of predilection for cerebral hemorrhages is the region of the central ganglia; this is explained by the anatomical peculiarities of the arteries which supply this region. They are end arteries; they leave the larger branches at right angles. Changes in the eye grounds, particularly of the optic nerves, in cerebral hemorrhage are unusual. This is because the increased intracranial pressure following a hemorrhage rapidly becomes equalized. If the hemorrhage is large and situated at the base of the brain, it may enter the sheath of the optic nerves and thus produce either an optic neuritis or a choked disc. This form of cerebral hemorrhage does not occur in cerebral apoplexy and results from rupture of an aneurysm at the base or from meningeal hemorrhages; basal hemorrhages, on the other hand, with entrance of blood into the optic-nerve sheath are frequent in fractures of the base of the skull.

Uhthoff found optic neuritis in about 4 per cent. of his cases of cerebral hemorrhage. If optic neuritis be present it may be but another manifestation of the general cause which produced the hemorrhage of the brain (cardio-vascular lesions, nephritis).

Choked disc is somewhat more frequent; it was present in 7 per cent. of Uhthoff's statistics, and in only a small part of the cases was there a hematoma of the optic nerve sheath. The most probable explanation of this is that the papillœdema is due to increased intracranial pressure, and in this collateral inflammatory processes about an extravasation of blood may be a factor. In most cases the increased pressure is rapidly equalized and death often supervenes before the ophthalmoscopic condition develops.

Retinal hemorrhages are seen in cerebral hemorrhages more frequently than in cases of softening of the brain. Here again the fundamental condition—nephritis, diabetes—must be considered. It is generally recognized that retinal hemorrhages may be the precursors for cerebral hemorrhages, though Uhthoff believes that the frequency of this association has been over-estimated and a parallelism between the retinal and cerebral arteries does not necessarily exist. Particularly, pathological changes in the brain arteries which lead to hemorrhage are present without any changes in the retinal arteries. Straub claims that the prognosis of retinal hemorrhages in arteriosclerosis, as far as life is concerned, is not particularly unfavorable before the fortieth and after the sixty-fifth year. The serious age is between forty-five and fifty years. In Geis' excellent paper,[1] based on Uhthoff's material, 67 cases of retinal hemorrhages due to arteriosclerosis, diabetes, chronic nephritis, and without discoverable cause were followed in practically all of the cases in subsequent years by an affection of the brain (hemorrhage, softening, etc.). This complication usually occurred in from one to two years, occasionally not before the fifth or sixth year. The retinal hemorrhages were frequently accompanied with increased blood pressure and this association is particularly ominous. Increased blood pressure is generally absent in retinal hemorrhages which are due to anemia, sepsis, etc.

The prognosis of cases with so-called apoplectic retinitis (thrombosis of the central retinal vein) is quite different, and in only 50 per cent. of the cases were cerebral complications observed after a number of years. The most favorable prognostic significance as regards cerebral complications are retinal hemorrhages which are due to an altered condition of the blood (hemophilia, anemia, sepsis, etc.). Recurring conjunctival hemorrhages and vitreous hemorrhages are also without grave significance.

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[1] Geis, Kl. M. f. A., Vol. XLVIII, 1910, p. 495.

Homonymous hemianopsia is the only form of hemianopsia seen in cerebral hemorrhage and the affection is then central to the large basal ganglia. The usual site is in the neighborhood of the internal capsule where the eye changes are complicated with hemiplegia and hemianæsthesia of the opposite side. In the optic radiation as well as in the cortical and subcortical visual areas, hemorrhages producing homonymous hemianopsia are much more unusual. In this region embolic and thrombotic processes are more common. Uhthoff found hemianopsia in 35 per cent. of his cases of cerebral hemorrhage with eye symptoms; the hemianopsia was complete in 23.8 per cent., partial in 4 per cent., color hemianopsia in 1.9 per cent., and bilateral in 5.6 per cent. Gowers has particularly emphasized the frequency of hemianopsia occurring directly after an apoplectic stroke in combination with hemiplegic symptoms and conjugate deviation of the eyes and of the head. Uhthoff believes that the frequency of this transient hemianopsia is somewhat exaggerated, in consideration of the difficulty of a functional examination in the patients with this lesion. Motor hemiplegia is generally associated with pronounced disturbances of sensation in the same half of the body. Infrequently the hemianopsia is combined with only a disturbance of sensibility, without hemiplegia. Hemianopsia is much more frequently combined with both motor and sensory disturbances. Isolated hemianopsias without involvement of the body are more apt to be due to embolic and thrombotic processes than to cerebral hemorrhages. The transcortical disturbances of vision (mind-blindness, alexia and optical aphasia) are very infrequent in brain hemorrhages and suggest a softening process due to either embolism or thrombosis.

Conjugate deviation of the eyes is the most frequent disturbance of ocular motility in cerebral hemorrhage. Uhthoff found it in 28 per cent. of his cases and believes that these figures are too small, particularly as the symptom is apt to be a transient one. In recent hemorrhage it is unquestionably more frequent. Wernicke[1] distinguishes between paralysis of the associated lateral ocular muscles (in pons disease) and conjugate deviation. The conjugate deviation in hemiplegia he regards as due to the overpowering influence of one hemisphere, and if the patient has not lost consciousness the eyes can be brought to the middle line. The deviation of the eyes is often lost as the coma disappears. The localization of the conjugate deviation is still unsolved and it seems improbable that this symptom depends upon a lesion of a definite part of the brain, because it has been observed in hemorrhages in all parts of the brain. At the same time, its appearance indicates a large extravasation of blood and is generally associated with coma.

Isolated paralysis of an ocular muscle in hemorrhage of the brain is very infrequent. This is explained by the fact that a supranuclear or cortical disturbance of the ocular muscles affects the associated or the disassociated movements of the eyes. Hemorrhages in the peduncles or pons, in the neighborhood of the fourth ventricle or in the corpora quadrigemina may produce nuclear or fascicular paralysis of the ocular muscles. The only isolated disturbance of ocular movement which is due to a cortical lesion is the so-called cortical ptosis; this is occasionally seen in hemorrhage of the brain, though it is more frequent in softening and in tumors. Ptosis with paralysis of the opposite half of the body indicates a lesion of the peduncle. Paralysis of the oculomotor nerve in its various branches is very unusual. This also holds for all the other disturbances of the ocular muscles. Changes in the pupils are inconstant; they are sometimes dilated and in other cases they are contracted, so that they do not offer any diagnostic aid.

Softening (Encephalomalacia).—After complete ischemia, which is the pathological process in softening, the brain substance undergoes edematous swelling and finally complete necrobiosis, the latter occurring particularly when endarteries are affected.

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[1] Quoted from Uhthoff l. c.

The eye symptoms are more directly due to local changes than to distant action, as is seen in hemorrhages. In softening of the brain ophthalmoscopic changes particularly of the optic nerves, are comparatively unusual. This is due to the same conditions which govern eye-ground changes in cerebral hemorrhage, the factor of intracranial increased pressure is even here of less significance, though the pathological changes of the eye grounds may again be due to associated pathological conditions (cardio-vascular disease, nephritis, diabetes, etc.). Optic neuritis was observed in softening following embolism or thrombosis in 6 per cent. of Uhthoff's cases. Choked disc, on the other hand, was practically not observed; the same holds true for optic atrophy.

The clinical picture of occlusion of the central retinal artery as it occurs in embolism or thrombosis is unusual in softening of the brain (about 2 per cent. of the cases). Just as in occlusion of the retinal artery a thrombotic process is much more frequent than an embolic one, the same relationship holds true in softening of the brain. A closure of the internal carotid artery does not cause any change in the ophthalmoscopic picture if the anterior and posterior communicating arteries are intact. A sudden closure of the carotid is followed by a transient reduction of the arterial current in the eye. At the same time pronounced sclerotic changes in the retinal vessels or a thrombosis of the central retinal artery is of unfavorable prognostic significance on the state of the cerebral arteries. Though sclerotic changes in the arteries of the brain occur more frequently than in the retinal arteries, if these changes are evident in the retina, they are always present in the cerebral arteries. Geis[1] has shown that 17 patients with pronounced sclerotic changes in the retinal arteries died within four years with the symptoms of a brain disturbance

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[1] Geis, Kl. M. f. A., 1910.

(apoplexy), which all seemed to be due to softening and usually were fatal. Thirty-five cases of closure of the central retinal artery showed in their subsequent course that 17 suffered from an apoplectic stroke, usually in from one to two years later, though in some this complication did not occur until the sixth year. These patients were all over forty years of age and did not present any marked cardiac lesion. The cases of embolic closure of the retinal artery observed in adolescents with a cardiac lesion give a better prognosis for a possible brain complication. The conditions in the cases of thrombosis of the central retinal vein are different. Forty-four cases were observed for more than five years. Of these, 20 subsequently suffered from an apoplectic attack, usually in from one to two years later; others not before the ninth, tenth and twelfth year. The remaining 24 were still in good health in from five to twelve years after the eye lesion. This shows that in over 50 per cent. of the cases the change in the retinal vein was purely a local one and a complicating disease of the cerebral vessels could not be assumed. If in these cases of venous thrombosis there were also found distinct arterial changes in the retina, the prognosis was, of course, much more unfavorable. Pronounced sclerosis of the choroidal vessels do not permit any deductions on the condition of the cerebral vessels. In brain syphilis, syphilitic endarteritis of the cerebral arteries is frequent. At the same time, similar changes in the retinal vessels were very rarely observed. This again shows that the retinal vessels in general are much less frequently affected than the cerebral vessels. A closure of the retinal artery is usually not accompanied with large retinal hemorrhages. The so-called hemorrhagic infarct with numerous hemorrhages does not belong to the clinical picture of an arterial occlusion in the retina, but always indicates an associated pathological change in the retinal vein. Isolated retinal hemorrhages without neuritic or retinal changes are uncommon in softening of the brain.

The most frequent symptom of softening of the brain is homonymous hemianopsia, as far as the ocular symptoms are concerned. This was observed in 51 per cent. of Uhthoff's cases. The homonymous hemianopsia was complete in 36.4 per cent. partial in 3.5 per cent., and bilateral in 11.4 per cent. The cause for homonymous hemianopsia in cerebral softening was practically never due to a lesion of the optic tract. In 22 per cent. the areas of softening were found in the region of the primary optic ganglia, that is, in the beginning of the optic radiation near the internal capsule; and in 77 per cent. of the cases the optic radiation and the visual cortical region were involved. In cerebral hemorrhage with homonymous hemianopsia this proportion is different: in 58 per cent. the optic paths were affected centrally from the primary optic ganglia, while in 42 per cent. the optic ganglia themselves were involved.

If the homonymous hemianopsia is due to a lesion of the primary optic ganglia or the very beginning of the optic variation, the so-called **macular preservation** of the visual fields in the defective halves was not observed. Uhthoff says that a macular preservation requires a preservation of at least 5° to 10° beyond the point of fixation. Small defects of 1° to 3° can not be regarded as true preservation and are probably errors in the field examination. In cases of softening in the posterior parts of the optic radiation and in their cortical termination the hemianopsia presented a macular preservation with such regularity that this may be regarded as the rule. According to Lenz,[1] macular preservation occurs in cortical and subcortical involvement of the optic paths hemianopsia in 83 per cent. of the cases. On the other hand, in affections of the primary optic ganglia and in the beginning of the optic radiation as well as affections of the tract and of the chiasm the dividing line of the hemiopic visual-field defect passes directly through the point of fixation. The boundary line between the frequent presence of macular preservation and its absence corresponds to the middle third of the parietal lobe. A lesion of the internal capsule with hemiplegic symptoms is generally not accompanied with hemianopsia. This complicating eye symptom is observed only if the lesion is deeply placed and extends to the optic pathways.

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[1] Quoted from Uhthoff, Graefe-Saemisch, Vol. XI, 3, p. 976.

Hemiachromatopsia was found in about 6 per cent. of Uhthoff's cases. Bilateral hemianopsia is much less frequent than monolateral hemianopsia in softening and hemorrhage. Uhthoff mentions the proportion of the bilateral to the monolateral as being 1:13. Subjective sensation of light and visual hallucinations are most frequently observed in cerebral softenings with hemianopsia; the lesion was always in the optic radiation. Hemianopic hallucinations are not infrequent in diseases of the occipital lobe, and are usually present in the blind halves of the field. Alexia (word-blindness) as a striking symptom was found by Uhthoff in 32 out of 368 cases of brain softening where an autopsy had been performed. The word-blindness was present without hemianopsia in 6, with right-sided hemianopsia in 25, and with bilateral hemianopsia in 1. The lesions at autopsy were found grouped together about the region of the angular gyrus and along the inner surface of the visual sphere extending into the medulla. A simultaneous hemianopsia is observed with alexia the further back and nearer to the inner surface of the occipital lobe the lesion is situated. The lesion in the cases without hemianopsia were situated along the outer surface (convexity) of the occipital lobe. Mind-blindness was observed in the above-mentioned series in 21 of 368 cases of brain softening. This symptom occurred in 7 without hemianopsia, in 4 with right hemianopsia and in 14 in bilateral hemianopsia. Most of the lesions were situated at the inner surface of both occipital lobes. Mind-blindness occurs more frequently in brain softening and in more than one-half of the cases it is associated with hemianopsia, usually bilateral. The ophthalmoscopic condition in hemianopsia following softening and hemorrhage is generally negative.

Paralysis of the various ocular muscles is a very unusual symptom in softening of the brain. The oculomotor nerve was affected in 2.4 per cent., the abducent in 0.3 per cent. of Uhthoff's cases, and there were no cases of trochlear involvement. These numbers are so much smaller than those found in hemorrhages of the brain, as is readily apparent on considering the character of the pathological lesion, that a pronounced ocular muscle lesion, if disturbances of associated lesions and those due to affections of the peduncles, pons and corpora quadrigemina are excluded, is against the diagnosis of a softening process.

Conjugate deviation is not observed in affections of the frontal and the occipital lobes. They seem to be caused by lesions about the central fissure, namely, the anterior and posterior central convolutions. Cerebral hemorrhages are a frequent cause for this conjugate deviation when situated in the region of the large central ganglia. As a rule in diseases of the cerebrum the patient usually has his eyes turned to the side of the lesion. Disturbances of the pupil are not characteristic.

Sclerosis of the Cerebral Arteries.—In pronounced sclerosis of the cerebral vessels distinct pathological changes have been observed in the eye grounds in a number of cases. These changes consist in abnormal tortuosity and irregularity of the arterial lumen, white sheathing of the vessels, whitish degeneration and a clouding of the vessel wall, and thrombosis of the retinal artery. In fact these ophthalmoscopic changes, if present in both eyes, furnish us with important information on the condition of the cerebral vessels, particularly of those participating in the distribution of the internal carotid.

It is remarkable how frequently no changes were to be seen in the retinal vessels with the ophthalmoscope in pronounced sclerosis of the cerebral arteries confirmed by autopsy. Hertel has drawn attention to certain pathological changes in the retinal vessels as being purely senile changes and that these naturally do not suggest an angiosclerosis of the vessels of the brain. Uhthoff takes a distinctly conservative attitude and does not believe that slight ophthalmoscopic changes, such as a bright vascular reflex, tortuosity of the vessels, slight variations in calibre, signify a pronounced pathological change in the retinal vessels, and he is of the opinion that certain authors—Raehlmann, Gunn and others—have gone too far, he particularly does not confirm Gunn's statement that a serious prognosis for the general vascular system is present when there are these slight changes in patients between forty and fifty years of age.

A negative ophthalmoscopic condition of the retinal vessels does not indicate a normal condition of the cerebral vessels; even in marked arteriosclerosis of the carotid artery and of the ophthalmic artery the central retinal artery, in many cases, has been normal. Sclerosed cerebral arteries sometimes injure the optic nerves and the basal optic tracts through compression. A number of investigations have shown that the sclerosed internal carotid or the ophthalmic artery can produce a pressure atrophy of the intracranial part of the optic nerve.

Some authors have found a more or less concentric contraction of the visual field in general sclerosis of the cerebral arteries. The general symptoms in these cases were more or less vague and consisted in headache, vertigo, and a diminution in mental ability. The concentric contraction of the field was even regarded as an important symptom in the diagnosis of general cerebral sclerosis. In a number of cases of patients between seventy and eighty-three with pronounced atheroma of the cerebral arteries, Planck was not able to confirm the presence of this contraction of the field and Uhthoff is inclined to regard it as an instance of the functional concentric contraction which occurs in neurasthenia, etc.

Brain Tumor.[1]—INTRODUCTION.—Brain tumor is not a very unusual condition; it occurs twice as frequently in men as in women. Though it appears at any age, it is most frequent between puberty and thirty years and tumors in children are not infrequent. The most frequent tumors of the brain are glioma and sarcoma, in children the solitary tubercles are more frequent. Glioma is a tumor which develops only primarily, but is usually solitary. It is difficult to limit the growth macroscopically on account of its characteristic infiltrating tendency. This quality leads to a growth in between the nerve fibres, whereby their function is not destroyed until late. Glioma degenerates easily, usually in the form of cysts. Sarcoma develops from vessel walls, from the meninges and the bone, they originate therefore either in the brain substance or from the meninges or the bone. Cerebral sarcoma is a primary brain tumor. It is a realtively benign form of sarcoma, varying in size and differs from the glioma in its growth in tumor form; in other words, it compresses and displaces the brain substance without directly infiltrating it. Macroscopically a sharp limit between the tumor and brain substance can be recognized.

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[1] Bruns in Krause, Allgem. Chirurgie d. Gehirnkrankheiten, Part II, p. 19.
Uhthoff, Graefe-Saemisch, II ed., Vol. XI, p. 1143.
Uhthoff, Bowman Lecture T. O. S., 1914.

The infectious granulomata are usually tubercles. Tuberculous disease of the brain occurs in the form of miliary nodules in tuberculous meningitis or in circumscribed conglomerate foci, the so-called conglomerate tubercle. This is usually (*a*) a flat but extensive process, occurring in the soft membranes on the surface of the brain, which slowly invades the brain tissue. It produces the symptoms of a tumor if it is situated in regions capable of localization. Another form is (*b*) the so-called solitary tubercle, which is situated in the brain tissue, and is the most frequent form in childhood. The size differs enormously. It is especially apt to invade the cerebellum and the base of the brain, particularly the pons. In addition to brain tuberculosis, the lungs, the middle ear, the bones and the joints are involved. Surgically, the conglomerate tubercle is inaccessible. Syphilis appears in the form of flat meningoencephalitic processes on the surface of the brain. These are particularly amenable to anti-syphilitic treatment. The large intracerebral gummata do not yield to treatment so readily. The gummata are usually in the cortex on the convexity of the brain. This is possibly due to the rôle of trauma in their development. Their frequent site in the central convolutions is the reason why Jacksonian epilepsy is so frequently produced by gummata. The size of the brain gumma is usually small; it is rarely over the size of a walnut. The gumma shows a strong tendency to necrosis and caseation, and at autopsy it is often difficult to differentiate between a gumma and a tubercle.

General Symptoms.—The growth of a tumor in the brain produces increased intracranial pressure, due in part to an increase in the cerebro-spinal fluid and to internal hydrocephalus. Increased brain pressure causes clinically symptoms of disturbance of consciousness, headache, vomiting, slow pulse and choked disc. The internal hydrocephalus may produce local changes at the chiasm, when the floor of the third ventricle is pushed down. The basal cranial nerves are also pressed upon by the distended ventricles, especially the oculomotor and the abducent nerves. The action on the oculomotor nerve sometimes shows itself by isolated ptosis or mydriasis. Another factor in producing general symptoms is the so-called brain swelling. Thus small tumors may produce brain swelling which is absent in large tumors. This may explain the discrepancy in the degree of choked disc. Generally the occurrence and intensity of the choked disc depends on the site of the tumor. The general symptoms are due to the conditions which lead to increased pressure, to hydrocephalus and to brain swelling.

The local symptoms depend upon the part of the brain in which the tumor is situated. The action of a brain tumor is divided into three stages: irritation, compression and destruction. Thus following Jacksonian attacks, monoplegia develops and hemianopsia comes after visual irritations. The general symptoms can be described as headache, vomiting, vertigo, psychic disturbances, general convulsions and choked disc. The local symptoms are: paralysis, disturbance in sensation, ataxia, disturbance of special sense and of speech. The headache and choked disc are most pronounced in tumors of the posterior fossa, as these lead to early and pronounced hydrocephalus. This also is true for vomiting, slow pulse and vestibular vertigo. Certain symptoms are described as distant symptoms, which make a local diagnosis often impossible. The importance of these symptoms has been exaggerated and at the same time their occurrence can not be denied.

The headache in brain tumor is the most constant and the most important general symptom. It is a dull, deep-seated pain which somewhat resembles migraine, often associated with vomiting, sometimes pulsating in character. It becomes continuous and occasionally increases to an unbearable degree, and the patient loses consciousness. In children it is apt to be slight as long as the skull can distend. Vertigo is not a frequent symptom; the patient has the sensation that the objects in the room rotate about him and that his body seems involuntarily to be turning in one direction. The appetite is not lost and does not turn on the taking of food. Nausea is frequently absent and the vomiting has the characteristics of projectile vomiting. The reduction of pulse rate is not a frequent nor important symptom of brain tumor. An important psychic disturbance is stupor. The patient at the beginning appears like one asleep. This symptom increases so that the patient will fall asleep while eating. The convulsions, if they are general like in classical epilepsy, are regarded as general symptoms. The so-called Jacksonian convulsions are a definite focal symptom in diseases of the anterior central convolutions. When epileptic convulsions occur in later life, a tumor must always be suspected and other symptoms looked for. The most important symptom is choked disc. It is the objective sign of an organic brain disease. All other symptoms may occur in functional affections.

Ocular Symptoms.—Ocular symptoms which help in localizing brain tumors may be the expression of local or of distant action of the brain tumor; it is often difficult to distinguish between these.

The most frequent change of the eyes in brain tumor is a lesion of the optic nerve which occurs in the form of papillœdema, optic neuritis, neutitic atrophy, or simple atrophy.

The most recent explanation for the development of a choked disc is given by Schieck,[1] who believes it to be the result of increased pressure and an abnormal accumulation of spinal fluid in the intervaginal space following an increase of intracranial pressure which, in turn, interferes with the lymph current returning from the vitreous and from the disc centripetally in the perivascular lymph spaces contained in the axial bundle. Schieck found the lymph spaces in choked discs always dilated and the central vein in the optic nerve compressed. This would prove that choking of the disc is at first a mechanical process and that the inflammatory symptoms are secondary. Kampherstein[2] in Uhthoff's clinic examined 55 eyes from 44 cases. He found a hydrops of the intervaginal space in 65 per cent. of the cases. The dilatation was most marked next to the eye ball and generally between the arachnoid and the pia. In 76 per cent. of the cases there were inflammatory changes in this vaginal space which were more marked the older the process. In 35 per cent. of the cases there was no dilatation of this space and no inflammatory symptoms, which show that a choked disc may occur without these changes.

According to Paton and Holmes,[3] both factors in papillœdema, the venous congestion and the obstructed lymph drainage, are due to pathological increase of the pressure in the optic sheath. Paton had not found arching forward of the lamina, and was not in favor of choked disc being the result of venous congestion alone. There must be a second factor in the shape of the blocking of lymph drainage.

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[1] Schieck, Die Genese der Stauungspapille, Bergmann, Wiesbaden, 1910.

[2] Kampherstein, Kl. M. f. A., 1904, 1905.

[3] Paton and Holmes, Pathology of Papillœdema, T. O. S., 1911.

As the intraocular and the intracranial pressures are normally the same, Henderson[1] states that choked disc is a manifestation of a disturbed equilibrium between these two. When the intracranial pressure rises the cerebral venous pressure rises and in the retinal vein. The arching forward of the lamina and so-called hydrops represents a yielding of those structures to the increased hydrostatic pressure. As the brain acts as viscous mass the pressure in one hemisphere and corresponding optic nerve may rise above that of the other, thus acounting for the ipsolateral feature of choked disc.

Schieck[2] finds that the primary factor is the infiltration of the peripheric parts of the optic nerve with cerebrospinal fluid, thus causing a congestion of the central vein which leads in turn to an edematous swelling of the disc. This author has made injections into the sheath of the optic nerve in cadavers and has produced characteristic choked disc.

Uhthoff[3] does not believe that a choked disc can develop from primarily an inflammatory process. The inflammatory changes in the optic sheaths and at the disc do not belong to the anatomic picture of choked disc and are absent in a number of cases. They are, therefore, better regarded as secondary, following degenerative process of the optic nerve. It has never been possible to discover toxins or phlogogenic material and attempts to produce inflammatory changes in animals' eyes with the spinal fluid from cases of tumor of the brain have not been successful.

The increased intracranial pressure in brain tumor, usually accompanied with hydrocephalus, surely plays the main rôle in the development of choked disc. The hydrops of the sheath is a frequent symptom, though not absolutely necessary. Choked disc usually means an elevation of 1½ to 2 D. The clinical condition of beginning "choked disc" can be diagnosticated when distinct ophthalmoscopic changes are present with unchanged sight and field or with typical periodic obscurations. Uhthoff believes that in the early changes the mechanical factor (increase of intracranial pressure with internal hydrocephalus) is the effective cause. The pronounced inflammatory symptoms in the later stages are secondary.

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[1] T. Henderson, The Pathogenesis of Choked Disc, T. O. S., 1912.

[2] Schieck, Die Bedeutung d. Stauungspapille, M. m. W., 1913, No. 1.

[3] Uhthoff, *l. c.*, p. 1149.

v. Hippel[1] pleads for the importance of a recognition of choked disc in its earlier stages. In the beginning it is not an inflammation and must be sharply opposed to primary inflammatory processes; after long existence inflammatory changes may be added. V. Hippel, among others, furthermore does not believe the limit of 2 D. is necessary to determine choking, if it can be carried through practically. In cases of less swelling much information can be obtained from a more careful ophthalmoscopic examination, from the functional examination, from the general symptoms and clinical course. According to Schieck,[2] the first change is a haze over the middle of the papilla where the large vessels emerge. In the upright image this is anterior to the detached limitans. Soon an exudate appears which projects into the vitreous. Bright lines (lymph spaces) appear, a linear cloudiness accompanies the vessels and adjoining retina, the disc margin becomes blurred wherever the vessels travel and the greatest elevation corresponds to these vessels. Horsley[3] states that the choked disc always begins in the upper and nasal quadrant, then extends to the lower nasal and finally to the lower temporal. v. Hippel found that the large Gullstrand binocular ophthalmoscope gave a remarkably clear picture of the protrusion of the disc. These pictures only denote beginning choked disc when the functional examination is negative, except for enlargement of the blind spot and general symptoms such as headache, vomiting, etc.

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[1] v. Hippel, Ueber die Bedeutung d. Stauungspapille, Centralbl. d. Ges. Ophth., 1914.

[2] Graefe's Archiv, Vol. LXXVIII, p. 1.

[3] B. M. J., 1910.

An ophthalmoscopic picture which is sometimes difficult to correctly interpret is pseudo-neuritis.

In *congenital pseudo-neuritis* the disc is red, the margins are completely blurred, the vessels are congested and tortuous and the disc can even be somewhat prominent. At the same time these changes may all be physiologic. This congenital anomaly is generally found in hyperopia and astigmatism, the addition of colloid excrescences (whitish yellow spots at the margin of the disc) still further obscures the picture. As this refractive condition is often accompanied with headache and vomiting, the thought of a tumor occurs. If the greyish opacity of the surrounding retina or the smallest hemorrhage is added to the above described picture, it immediately becomes pathologic. In the diagnosis of incipient optic neuritis it is very important to observe whether this opacity is present in the retina and particularly if there are any hemorrhages. Uhthoff mentions two cases of congenital pseudo-neuritis[1] with violent cerebral symptoms which consequently offered difficulty in diagnosis, and later proved to be hysteria.

Various statistics have shown the **frequency of choked disc and optic neuritis** in tumors of the brain to be 80°. Uhthoff found the occurrence of choked disc to be:

	Per cent.
Brain tumor	74.6
Cerebral syphilis	12.0
Brain abscess	2.2
Internal hydrocephalus	2.2
Serous meningitis	2.2
Tower skull	2.2
Meningitis	1.1

Non-intracranial diseases produce choked disc as follows:

	Per cent.
Nephritis	1.1
Lead poisoning	0.3
Anemia	0.9

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[1] An excellent illustration of pseudo-neuritis accompanies Uhthoff's Bowman Lecture, 1914.

The optic nerves seem to be comparatively free from involvement when the brain tumors are situated in the motor region of the cerebral cortex, on the convexity of the hemispheres, and in the corpus callosum. It is also unusual in tumors at the base of the skull to produce choked disc and optic neuritis, and Seligmann believes that while tumors at the base of the skull produce paralysis of the cranial nerves, a normal ophthalmoscopic picture is the rule. Gowers has shown that tumors arising from the meninges and exerting compression of the brain without infiltration lead to changes in the discs less frequently than those which grow in the substance of the brain, and if the tumor is compressing the brain the symptoms will depend upon the rapidity in which its growth takes place.

The tumors in the pituitary region with or without acromegaly are also different. Optic neuritis or choked disc was present in only 20 per cent. of the cases. Simple optic atrophy is here the preponderating ophthalmoscopic change. It is generally recognized that tumors of the cerebellum and of the posterior cranial fossa cause choked disc and optic neuritis more frequently than other tumors of the brain (88 per cent. of the cases). The tumors of the pons, produce inflammatory changes of the disc in 70 per cent. according to Uhthoff.

The most important factor in the differential diagnosis of tumors is the relatively frequent presence of a simple atrophy of the optic nerves in tumors of the hypophysis, as compared to tumors in other regions where a simple atrophy is very exceptionally found present (1.1 per cent.). The steep protrusion of the clouded papilla is the most characteristic change in the ophthalmoscopic picture of choked disc. The prominence must be at least 2 D. The changes about the optic nerve are sharply limited from the rest of the retina. Small white areas and hemorrhages in the region of the clouded disc are not unusual.

Other whitish retinal plaques and the star-shaped figure in the macula are unusual, though it has been repeatedly observed that the ophthalmoscopic picture in brain tumor may resemble that of albumenuric retinitis, though even in these cases the steep and prominent choked disc differs from the neuroretinitis seen in albuminuria. Paton[1] does not regard these macular changes as identical with those found in albumenuric retinitis, as different layers of the retina are affected, and the process indicates an intense œdema.

There are exceptional cases of chronic nephritis where the typical picture of choked disc without other changes in the retina are present and this picture could be easily confounded with that seen in brain tumor. Large and massive hemorrhages about the disc are exceptional.

The disturbance of vision in choked disc has a number of characteristic features. Thus, sight may remain unaffected for a long time with pronounced ophthalmoscopic changes. Secondly, periodically recurring transient obscurations are of direct diagnostic value and presumably indicate variations in the intracranial pressure (distension of III ventricle).

The field may be changed in a number of ways. Aside from an enlargement of the blind spot, a concentric contraction is probably the most frequent. Hemianopsia and central scotoma are not frequent. Cushing and Heuer[2] found in 123 brain tumors that 53 showed crossing or inversion of the color fields. In 10 this dyschromatopsia preceded the ophthalmoscopic signs or occurred in the earliest stages of choked disc, hence the importance of careful perimetric examination. Leslie Paton[3] observed a central scotoma in a frontal tumor pressing on one optic nerve; optic neuritis in the other eye, while primary optic atrophy was present in the affected eye. Attention to one-sided choked disc or optic neuritis in brain tumor has been particularly drawn by Horsley, who believes that a one-sided ophthalmoscopic picture with great probability indicates the side of the tumor. Horsley[1] states that homolaterality of the degree and age of the neuritis are of great clinical value. The age of the swelling is of as great importance as the mere engorgement of the disc. This is naturally a question of the greatest importance for operation. A number of observers have controverted this statement; a one-sided choked disc or a choked disc more pronounced on one side than on the other, is of certain evidence that that is the side of the tumor, though this factor is not by any means definite. Mohr[2] has investigated the cases in Uhthoff's clinic and has found 41 cases of one-sided choked disc; in 23, that is, 56 per cent., it was on the same side as the tumor, and in 18, that is, 43 per cent., it was on the opposite side. Paton[3] in 50 per cent. found no difference in elevation of the choked disc on the two sides. In 25 per cent. the swelling was greater on the side of the tumor, 25 per cent. it was greater on the opposite side.

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[1] Paton, Optic Neuritis in Cerebral Tumors, T. O. S., 1908.

[2] Cushing and Heuer, Distortion of the Visual Fields in 200 Brain Tumors, Sec. Ophth. A. M. A., June, 1911.

[3] Leslie Paton, T. O. S., 1910, p. 133.

One-sided optic neuritis in brain tumor in nearly all of the cases was on the same side as the tumor. In bilateral optic neuritis or choked disc in which one side was more marked than the other, the side of the most marked fundus change was on the side of the tumor in 72 per cent. In cases of atrophy of one side with choked disc on the other, the choked disc in all of the cases was on the other side from that of the tumor. Parker[4] and Ley[5] have endeavored to trace the varying intensity of the choked disc to the intraocular pressure, and that the lesser prominence was present in the eye in which the intraocular pressure was greater.

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[1] Horsley B. M. J., July 25, 1909.

[2] Mohr, Kl. M. f. A., p. 401, 1910.

[3] Paton, T. O. S., 1908.

[4] Parker, J. A. M. A., 1916. T. A. O. S., 1911.

[5] Ley, B. M. J., 1910, p. 919.

The ophthalmoscopic changes at the optic disc do not give definite information either on the localization, the composition or the size of the tumor of the brain. In general, slowly growing tumors are less apt to cause choking of the disc than those that grow rapidly; at the same time, relatively small cerebellar tumors may early lead to a choked disc wherein the principal factor lies in the associated hydrocephalus. Simple optic atrophy is an extremely unusual ophthalmoscopic finding and, if it be bilateral, speaks against the diagnosis of tumor of the brain. The cases of tumor which are situated basally and which directly compress one of the optic nerves, have been known to produce a choked disc in the second eye; these are generally situated in the anterior cranial fossa.

Hemianopsia occur in tumors of the brain in 18.2 per cent. of the cases, according to Uhthoff. The homonymous variety is most frequently observed when the tumor is situated in the occipital lobe. In these cases visual hallucinations, alexia, optic aphasia and mind-blindness are present. Homonymous hemianopsia is also produced by pressure upon the optic tracts. The tumors in this case are either situated at the base in the middle cranial fossa or at some distance away. In these cases optic neuritis or choked disc is frequently present as well as involvement of other basal cranial nerves. In about 10 per cent. of the cases the hemianopsia is produced by a tumor situated in the region of the primary optic ganglia and of the intracerebral optic conducting paths. In these cases there is generally associated hemiplegia.

Bitemporal hemianopsia is produced exceptionally by internal hydrocephalus, where distention of the floor of the third ventricle presses upon the chiasm. Tumors which press upon the chiasm or in the part of the optic nerves directly anterior to it have in some instances caused a central scotoma (Nettleship and Leslie Paton). Blindness in one eye with the ophthalmoscopic picture of atrophy and the gradual loss of the temporal half of the field in the second eye is an important symptom-complex, indicating a process localized in the anterior cranial fossa in which one optic nerve and the chiasm are involved.

Paralysis of the Ocular Muscles.—In Uhthoff's statistics muscular paralyses were found in 34 per cent. Twenty-five per cent. represent true paralysis of the ocular muscles, while in 9 per cent. there were changes in the associated movements and nystagmus. The VI and the III nerves are affected in about the same degree of frequency. The trochlear nerve generally escapes, unless it is involved together with other ocular nerves producing a more or less complete ophthalmoplegia. The isolated paralysis of the VI nerve is generally a distant symptom in increased intracranial pressure. No information is furnished as to the side of the tumor by this monolateral abducent paralysis. The combination of a one-sided or a bilateral abducent paralysis with bilateral optic neuritis or choked disc always suggests a brain tumor. The combination of a VI and III nerve paralysis usually indicates a basal situation of the lesion. In these cases other cranial nerves are also involved, and the condition is often a metastatic tumor at the base of the skull. The III nerve is involved in a variety of ways and to a varying degree.

Paralysis of associated lateral or vertical movements and of convergence are rarely symptoms of tumors of the brain and indicate an involvement of the posterior cranial fossa with pressure on the pons or the corpora quadrigemina. Conjugate deviation and nystagmus are unusual symptoms and of but slight diagnostic importance.

The **pupils** in tumors of the brain offer us but few points in diagnosis. The V nerve is rarely involved by tumors in the substance of the brain and its affection indicates the seat of the tumor at the base of the brain, when other basal nerves are usually affected. The same holds good for neuro-paralytic keratitis.

Pronounced monolateral exophthalmus in tumors of the brain usually indicates a direct involvement of the orbit. It may possibly be produced by the increased intracranial pressure with compression of the cavernous sinus. The involvement of the olfactory nerve is unusual in tumors of the brain.

Tumors of the Cerebellum.—It is generally claimed that the cerebellum and the posterior cranial fossa are frequent sites for brain tumors, particularly in children, where the tumor is usually a solitary tubercle. Tumors in the cerebellum generally present disturbances of equilibrium, vertigo, vomiting, atactic muscular movements in the arm and leg, absence of disturbance of sensibility and important ocular symptoms. Hemianopic disturbances are absent. Choked disc, optic neuritis or neuritic atrophy are extremely frequent in tumors in the posterior cranial fossa. They represent 88 per cent. of the cases in Uhthoff's statistics. This frequency has been confirmed by all investigators and is due to the anatomic relations of the cerebellum which cause a compression of the fourth ventricle and of the Sylvian aqueduct with a stasis of the spinal fluid in the third and lateral ventricles. Parinaud was one of the first to draw emphasis to the nearly constant association of internal hydrocephalus with choked disc. The one-sided change in the optic nerve gives us even less information on the side of the tumor in cerebellar lesions than it does in tumors of the cerebrum.

As is natural, disturbances in the ocular muscles in cerebellar tumors are very much more frequent than in tumors of the brain. While in tumors of the cerebrum the III nerve is most frequently involved, in tumors of the cerebellum the VI nerve and the associated movements are usually affected. Nystagmus is also much more frequent. Isolated paralysis of the abducent nerve without involvement of the III or IV nerve was found in 14 per cent. and it was bilateral in 6 per cent. The one-sided abducent paralysis was on the same side as the tumor in 75 per cent.

of the cases. This isolated involvement of the abducent nerve seems to depend upon its anatomic course and the location of its nucleus in the floor of the fourth ventricle. The abducent paralysis is frequently accompanied by changes in the optic nerve which suggest a tumor of the cerebellum. The III nerve is much less frequently involved. This also holds for the IV nerve.

Paralysis of associated movement and conjugate deviation are of more importance and occur more frequently in cerebellar tumors than in cerebral tumors. Of these the lateral associated movements are the ones most frequently involved. Nystagmus and nystagmoid movements have been frequently reported and occur next in frequency to multiple sclerosis in affections of the nervous system with nystagmus.

The V nerve is not so often involved in cerebellar tumors as in tumors in the anterior and middle cranial fossæ. Tumors in this situation often affect the auditory nerve and in half of the cases the tumor was situated in the cerebello-pontine angle or was a tumor of the auditory nerve itself.

Brain Abscess.—CEREBRAL ABSCESS.—Most brain abscesses are situated in contiguity to the focus from which they arise and are in the majority of cases uncomplicated with meningitis or sinus thrombosis. The proportion of cerebral to cerebellar abscess is 3 to 1, and the otitic origin is the usual one.

The ocular symptoms in order of frequency are choked disc, optic neuritis, oculomotor or abducent nerve paralysis, homonymous hemianopsia, and sensory aphasia.

Choked disc was present in 23 per cent. of Uhthoff's statistics. Optic neuritis occurs about as frequently. In brain abscess papillœdema is much less frequent than in tumor of the brain and does not reach the same degree of development which is usual in the latter condition. In 13 per cent. the choked disc was present on only one side and then usually on the side of the abscess, though in a few of the cases it was situated on the other side. When there was a difference in the degree of the swelling of the nerve-head, the side of the greatest elevation corresponded in nearly all of the cases with that of the abscess. The changes in the eye grounds may increase for a time even after a successful operation has been performed without indicating any complication, though they have sometimes accompanied the development of a second abscess.

Optic neuritis was present in 21 per cent. In general one-sided optic neuritis or neuritis more pronounced on one side indicates the side of the lesion. *Optic atrophy* is extremely unusual in brain abscess. An extradural abscess produces fundus changes in a much smaller proportion of cases.

Homonymous hemianopsia occurred in 9 per cent. of brain abscesses and the abscess was situated, in one-half of the cases, in the occipital lobe and was generally not otitic in origin. When the abscess is situated in the temporal lobe, where it is otitic in origin, the hemianopsia is due to an involvement of the internal capsule with affection of the opposite half of the body, including the opposite facial nerve.

Motor Ocular Nerves.—The oculomotor nerve in cerebral abscess was the most frequently affected; Uhthoff found it affected in 19 per cent. It is unusual for the entire III nerve to be affected, certain branches only are involved. One-sided mydriasis was observed in 28 per cent., and it was frequently associated with ptosis. It seems that a partial III nerve paralysis is an important diagnostic feature in abscess of the temporal lobe on the same side. The abducent nerve is affected less frequently, in 10 per cent., usually on the side of the abscess, though rarely it is situated on the opposite side. The abscess is always otitic and is either situated in the temporal lobe or extradurally. Trochlear paralysis is a very unusual symptom. Conjugate ocular deviation was seen in 6.3 per cent.; the abscess was situated in the frontal or temporal lobe. Associated lateral paralysis was practically not observed. Nystagmus is a very unusual symptom in cerebral abscess, while in cerebellar abscess it is very frequent (42 per cent.). This symptom occurs more frequently in cerebellar abscesses than in any other nervous disease, multiple sclerosis excepted. It is a valuable point in differential diagnosis between cerebral and cerebellar abscesses, at the same time its relation to a coexisting internal ear lesion must not be forgotten.

Fifth nerve involvement is unusual. Sensory aphasia has been observed in 8 per cent. of the cerebral abscesses and always in those situated in the left temporal lobe. It is often combined with other disturbances of the aphasic symptom-complex, and is an important diagnostic symptom of temporal lobe abscess. Exophthalmus has occurred when there was a direct communication of the extradural abscess and the orbit. The pupils present no noticeable changes unless as instance of partial III nerve paralysis.

CEREBELLAR ABSCESS.—Abscess in the cerebellum occurs one-third as often as in the cerebrum. The otitic origin is even more frequent (80 per cent.) than in the case of cerebral abscess. The diagnosis of cerebellar abscess meets with difficulty because some of its symptoms (vertigo, vomiting, staggering, nystagmus) may be caused by an internal ear affection.

Choked disc was observed in 23 per cent. and optic neuritis in 22 per cent.; in other words, about as frequent as in cerebral abscess, and together showing an optic nerve lesion in nearly half of the cases. The one-sided or greater one-sided development generally was found on the side of the lesion. The oculomotor nerve is affected in 14 per cent. and the paralysis is only partial. The abducent is affected in 12 per cent., somewhat more frequently than in cerebral cases. Conjugate deviation was present in 6 per cent. Nystagmus is, as has been mentioned, a frequent symptom. It is twice as frequent on looking to the affected as to the normal side (as occurs in labyrinth lesions).

Cerebral Syphilis.—Syphilis of the nervous system, in addition to the so-called metasyphilitic diseases—tabes and paresis—is one of the diseases which most frequently lead to eye symptoms. Nonne found 85 cases of true syphilis of the nervous system in 5500 nervous patients, and in his experience true cerebral syphilis is about twice as frequent as tumor of the brain and as multiple sclerosis. The postmortem records in cerebral syphilis have shown that in over one-half of the cases some part of the ocular apparatus was involved and in 25 per cent. the changes were situated at the base of the brain, usually in the form of a gummatous meningitis involving the intracranial optic nerves and chiasm.

The eye symptoms in brain syphilis result from the following pathological processes: syphilitic disease of the cerebral vessels and syphilitic neoplasm, the so-called gumma. Gummatous meningitis is situated usually in the region of the chiasm and in the space between the peduncles, and may extend irregularly over the entire base of the brain. Gummatous neuritis affects in order of greatest frequency the basal optic paths (tract, chiasm and optic nerve) and the oculomotor nerves, though the other cranial nerves may also be affected.

Uhthoff found that in 40 per cent. of his cases **pathological changes** were present at the **disc,** consisting in (1) choked disc, (2) optic neuritis or neuritic atrophy, and (3) primary atrophy, all in about the same frequency. In 7 additional cases there were disturbances of vision in the form of hemianopsia without ophthalmoscopic signs, making an involvement of the optic paths in one-half of the cases. This percentage of optic-nerve involvement is greater than that found in multiple sclerosis and is probably surpassed in frequency only by brain tumor. Cerebral lues causes choked disc next in frequency to brain tumor; a tumor-like syphilitic neoplasm was generally present. In a part of these cases there were evidences of associated gummatous meningitis and disease of the blood vessels.

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Choked disc in cerebral lues may completely disappear, leaving an atrophic discoloration of the discs with comparatively good vision. Uhthoff thinks that this retrogression of a choked disc does not occur exclusively in syphilis but also in anemia, serous meningitis (hydrocephalus) and abscess of the brain.

The non-prominent optic neuritis or neuritic optic atrophy is observed somewhat less frequently than the choked disc. In 62 per cent. there was a basal syphilitic meningitis; in 38 per cent. a syphilitic neoplasm in part complicated with meningitis, disease of the blood vessels and encephalitis was present, as opposed to 65 per cent. in choked disc. Optic neuritis is not infrequently monolateral. The inflammatory changes at the discs may be slight and transient and are not always in proportion to the severity of the lesion in the optic nerve.

Simple optic atrophy with sharp margins and without signs of a preceding inflammation was somewhat less frequent than either of the preceding two and in the majority of cases was bilateral. Generally the disc was atrophic throughout its entire extent, though the nasal half often presented a slight pink tinge. Least frequent was an atrophy localized to the temporal halves of the discs as is found in toxic amblyopia and in multiple sclerosis. In about one-half of these cases a gumma was present, and in nearly all of the remaining cases a basal syphilitic meningitis was the cause. A simple atrophic discoloration of the discs indicating a primary gray optic nerve degeneration does not occur in cerebral syphilis. The optic atrophy is always a secondary one following an inflammatory process somewhat more centrally located in the optic paths; the changes in the visual field are also different.

According to Uhthoff's studies, the intracranial part of the optic nerve between the optic canal and the chiasm shows a distinct predilection for pathological changes in cerebral lues, consisting in pronounced inflammatory or true gummatous lesions. In the latter condition the optic nerve is enormously increased in volume. The pathological changes at the intracranial portion of the optic nerves were generally in combination with diseases of the chiasm or of the tracts; these cases all presented the picture of an extensive basal meningitis.

In the cases where the chiasm and the intracranial optic nerves are both affected, the starting-point is often in the chiasm, as is further suggested by the initial appearance of a bitemporal hemianopsia. The optic tracts are exceptionally involved, though it is not infrequent to have the anterior part of the tracts affected in diseases of the chiasm.

In addition to changes at the optic disc, other ophthalmoscopic changes in the eye grounds and evidences of previous inflammation in the anterior half of the eye were found in only a **small percentage** of the cases. These changes (uveitis, neuritis, retinitis, interstitial keratitis), though the expression of a previous syphilitic infection, were not in direct connection with the cerebral lesion.

Uhthoff found in his own cases that congenital lues was the cause for one-third of the cases of cerebral syphilis with associated peripheric changes in the eye ball. Patients with syphilitic disease of the eye ball do not as a rule subsequently develop cerebral syphilis. Motais[1] found that after an iritis the central nervous system became affected in 15 per cent. of the cases, the bones in 20 per cent.; after chorioretinitis, disease of the central nervous system occurred in 22 per cent. and of the bones in 8.3 per cent., while after neuritis the nervous system was exclusively affected. Motais believes that an involvement of the eyes always indicates a serious prognosis and demands intensive treatment of the syphilitic infection.

Pathological changes in the retinal vessels pronounced enough to be recognized with the ophthalmoscope, are usually absent, though pathological changes in the cerebral arteries in syphilis are frequent. Seggel's contention that syphilitic disease of the retinal arteries is a frequent, associated lesion of brain syphilis, does not seem correct. Syphilitic arteritis occurs in the retina and changes have also been described in the arteries in connection with chorioretinitis; it is, however, very unusual for cerebral disturbances to be preceded by changes in the eye grounds. Wilbrand and Staehlin found a hyperemic condition of the optic nerve in the early stages of syphilis which sometimes developed into a mild neuroretinitis. In Uhthoff's clinic a large number of cases in the early stages of syphilis were examined without confirming the presence of any symptoms of brain syphilis.

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[1] Motais, Rec. d'ophth., Juin, 1904, quoted from Uhthoff.

The most frequent **change** in the **visual field** in cerebral syphilis consists in homonymous or bitemporal hemianopsia. The basal origin for this change was usually shown by the associated involvement of other cranial nerves. Bitemporal hemianopsia, produced by a lesion of the chiasm, is more frequently due to brain syphilis than homonymous hemianopsia, which Uhthoff found in only about 10 per cent. of the cases to be due to syphilis. In brain syphilis complete loss of light perception, if the blindness be of short standing, need not be regarded as a hopeless symptom. The ophthalmoscopic changes of hemianopsia in two-thirds of the cases were those of an optic neuritis or atrophy, and in one-third were negative. Other, though infrequent, changes in the fields, according to Uhthoff, are concentric contraction, and a preserved excentric segment. Central scotoma indicating an involvement of the optic nerve anterior to the chiasm was but rarely observed, and is explained by the sheltered location of the papillomacular bundle in the center of the optic nerve.

It is well-known that the **ocular nerves** are frequently involved in cerebral syphilis. Schubert[1] found in 47 paralyses of the ocular muscles due to syphilis the oculomotor nerve was affected in 27, the abducent in 13, the trochlear in 1, and in 6 there were combinations of various

[1] Schubert, quoted from Uhthoff.

ocular muscles. In general the syphilitic involvement of the ocular muscles belongs to the later stages, usually after one to two years. Exceptionally they may occur much earlier. Wilbrand and Staehlin in recent cases of syphilitic infection found no ocular paralysis present in 200. Since the introduction of salvarsan ocular-muscle paralyses have undoubtedly been observed at an earlier stage than is usual. These early paralyses have been regarded as examples of neurorecidives, and have been cured with the continuation of mercurial treatment. These paralyses have thrown a new light on the pathological lesion and it seems probable that the syphilitic infection of the nervous system occurs at a much earlier stage than has been believed.

The **oculomotor nerve** is the nerve the most frequently involved; in Uhthoff's cases it was affected in 34 per cent., and the bilateral involvement is as frequent as the monolateral.

Bilateral oculomotor paralysis is an important symptom, as it occurs in cerebral syphilis more frequently than in any other intracranial disease. It was observed in 15 per cent. of Uhthoff's cases and was due to a syphilitic basal process, involving the interpeduncular space. Bilateral oculomotor paralysis is often complicated with lesions of other cranial nerves. The optic tracts were involved in the form of hemianopsias, both homonymous and bitemporal, in one-third of the cases; the abducent nerves were affected in 27 per cent.; the other nerves affected were the trochlear, trigeminal, olfactory and facial. Both the external and the internal ocular muscles supplied by the III nerve are generally involved, though symmetrical muscles in the two eyes may be alone affected, such as a bilateral ptosis or a bilateral involvement of the superior recti muscles.

The monolateral oculomotor affection without crossed paralysis is also due to a basal syphilitic process which in about one-third of the cases is the only cranial nerve affected. In the other cases it is complicated with lesions of the optic paths and of the other ocular motor and sensory nerves. In more than one-half of these cases all branches of the oculomotor nerve were involved.

Ophthalmoplegia interna as an isolated paralysis is unusual in oculomotor involvement in cerebral syphilis. This ophthalmoplegia is usually a nuclear lesion, consequently is not involved in pathological processes limited to the base of the brain. It occurs very much more frequently as an isolated lesion due to syphilis without cerebral complications or associated with tabetic or paralytic manifestations. It is sometimes a precursor of the latter conditions.

The typical periodic recurring oculomotor paralysis is not a symptom of brain syphilis. Monolateral oculomotor paralysis with crossed body paralysis is due to a lesion located at the base of the brain near the peduncle, and is often complicated by the associated involvement of other basal nerves.

Bilateral affection of the **abducent nerves** is a relatively frequent symptom in brain syphilis and the bilateral involvement occurs as frequently as the monolateral. The cause was always a basal process associated with involvement of other cranial nerves, such as the optic nerve, oculomotor, trochlear, and trigeminal. Abducent paralysis with hemiplegia of the opposite side is a very unusual symptom in syphilis. The lesion is always situated in the pons and in its posterior part. An involvement of the abducent nerve alone without affection of the other cranial nerves and a nuclear paralysis of this nerve are very unusual.

The **trochlear nerve** is involved in only a small number of cases, much less frequently than the optic, oculomotor or abducent nerves. It is never involved alone and its paralysis is frequently associated with that of the optic and abducent nerves.

Paralysis of the **trigeminal nerve** is comparatively frequent, it occurred in 14 per cent. of Uhthoff's cases, in other words, about as frequently as the abducent nerve. The cause was always a basal syphilitic process which involved other nerves, in order of frequency: optic, facial, oculomotor, abducent, etc. Neuroparalytic keratitis has been observed in more than one-third of the trigeminal affections in cerebral syphilis. It is generally associated with anæsthesia of the first branch of the trigeminal nerve.

Reflex iridoplegia with preservation of the convergence reaction is much more unusual in true brain syphilis than in the so-called metasyphilitic diseases. It is found in 10 per cent. of the cases of brain syphilis, while in tabes it has been observed in 60 to 90 per cent., and in paresis in 50 per cent. Brain syphilis may be complicated with tabetic or paralytic symptoms and reflex iridoplegia then depends upon one of the latter conditions. The total loss of pupillary reaction (light and convergence) was observed in 4 per cent. of Uhthoff's statistics. Isolated ophthalmoplegia interna is very unusual in this form of brain syphilis. It is generally an isolated lesion or one due to tabes or paresis. Reflex iridoplegia with optic neuritis suggests brain syphilis. An intermittent iridoplegia is a very unusual symptom which is probably more frequently found in brain syphilis than in any other condition.

Uhthoff has found that in about 15 per cent. of the cases of brain syphilis eye symptoms are absent. Hereditary syphilis was the cause of the nervous symptoms in a small percentage of the cases; the form of involvement did not differ from that found in acquired syphilis. Mention has been made of the comparatively frequent associated changes in the eyeball (iritis, interstitial keratitis, etc.), in nervous disease due to inherited syphilis.

DIFFERENTIAL DIAGNOSIS.—*Cerebro-spinal Syphilis and Other Nervous Diseases.*[1]

Tabes.—Tabes and cerebral lues are sometimes combined and the symptoms of both diseases occur conjointly. In tabes progressive optic atrophy is observed in 8 to 10 per cent. of the cases; the condition is always progressive and terminates in blindness. In lues bilateral blindness with the ophthalmoscopic picture of simple optic atrophy does not occur. In lues the atrophic discoloration of the discs is the result of a descending atrophic process from changes occurring further back in the optic pathways. This retro-ocular localization gives definite symptoms (field, relation of ophthalmoscopic picture to visual defect, frequent monolaterality, involvement of other basal nerves and of other cerebral focal symptoms). The frequent presence of hemianopsia, particularly temporal in character, does not belong to tabes. Optic neuritis and the symptoms of a retrobulbar neuritis are also foreign to tabes. Visual disturbances and ophthalmoscopic changes in cerebral lues are always accompanied by other cerebral symptoms, though these may be general in character, such as headache, vertigo. Isolated optic neuritis or a descending optic atrophy without other complications is very unusual in syphilis and particularly in brain syphilis; while tabetic optic atrophy may be for a long time the only symptom of this disease.

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[1] Uhthoff, Graefe-Saemisch, p. 1098, Vol. XI, 3.

Visual disturbances without ophthalmoscopic changes which are not uncommon in cerebral syphilis, do not occur in tabes.

The disturbances of the ocular muscles is also a point of differentiation. In lues they are more frequent and are complicated with an involvement of other cerebral nerves. The basal development of the ocular paralysis in cerebral lues is often characterized by bilaterality, particularly of the oculomotor nerves, involvement of all branches, pronounced paralysis combined with disturbances of vision, crossed hemiplegia and other cerebral symptoms, such as a paralysis of associated movements. In tabes the paralyses of the ocular muscles are not so frequent, 17 to 18 per cent.; they are incomplete, transient, and have the characteristics of nuclear paralyses—an isolated paresis of the inner or the outer ocular muscles as far as the III nerve is involved—sometimes external and internal ophthalmoplegia. The latter is particularly unusual in cerebral syphilis.

The pupillary symptoms are also different. Typical reflex iridoplegia with preserved convergence reaction and with miosis is comparatively unusual in brain syphilis, where the loss of reaction of the pupil to light is generally associated with a dilated pupil; the reaction on convergence and miosis are absent and other branches of the III nerve or other basal cranial nerves are affected with typical field defects (hemianopsia). The V nerve is involved with diminished sensibility in all its branches much more frequently in cerebral syphilis than in tabes; this also holds true for the neuroparalytic keratitis. The ocular symptoms of paresis correspond to those of tabes in comparison with cerebral syphilis.

Brain Tumor.—The clinical picture of a brain tumor may resemble that of brain syphilis, particularly as in brain syphilis a syphilitic gummatous tumor may be present. In brain tumor, however, extensive paralysis of the cranial nerves will be absent.

Choked disc is much less frequent in brain syphilis than in brain tumor. Involvement of the optic pathways, in the form of hemianopsia, retrobulbar neuritis, disturbances of vision without ophthalmoscopic change, or descending atrophy, and visual disturbances combined with multiple cranial nerve paralysis are much less frequent in tumor than in brain syphilis. Reflex or total iridoplegia with involvement of the accommodation and of other branches of the III nerve, which are relatively frequent in brain syphilis, are very unusual in brain tumor.

Multiple Sclerosis.—Multiple sclerosis and cerebro-spinal lues differ not only in the form of visual disturbance and in the ophthalmoscopic change but also in regard to the disturbances of the ocular muscles and of sensibility. In multiple sclerosis choked disc is exceptional, advanced optic neuritis is relatively unusual and is generally transient, and symptoms of a pronounced lesion of the optic pathways (hemianopsia, one-sided blindness) are absent. Bilateral blindness does not occur. The common visual disturbance consisting in a central scotoma, the incomplete atrophic discoloration of the optic nerve (temporal pallor), often in disproportion to the amount of visual defect, and the presence of a pathological optic nerve head without visual disturbance, are all symptoms peculiar to multiple sclerosis and point to lesions in the periphery of the optic nerve.

The frequent involvement of other cranial nerves combined with visual disturbances which are a common occurrence in brain syphilis are not observed in multiple sclerosis. A marked difference is also observed in nystagmus. Multiple sclerosis shows nystagmus present in 12 per cent. and nystagmoid movements in 46 per cent. Both of these are very infrequent in cerebral syphilis. The disturbances of the ocular muscles are also different. The ocular muscle paralysis in multiple sclerosis is characterized by being fleeting; a paralysis of associated movement is not infrequent, but complete paralyses of individual ocular nerves, and the involvement of other cranial nerves are absent.

In multiple sclerosis reflex iridoplegia does not occur. The pupils are rather narrower than normal and the reaction to light is prompt. The V nerve is also not involved in multiple sclerosis, while its affection in brain syphilis is frequent and often severe.

Hemorrhage and *softening of the brain* with their symptoms depending upon the part of the brain involved, if produced by syphilitic disease of the cerebral arteries, are frequently associated with other symptoms at the base of the brain and particularly with ocular changes resulting from basal meningeal processes. While isolated syphilitic endarteritis rarely occurs in the cerebral arteries with secondary hemorrhages and thromboses, as is seen in nonsyphilitic vascular changes in the brain, it is always complicated with other basal changes characteristic of brain syphilis. In apoplexy and softening isolated ocular paralyses of a basal or nuclear type and visual disturbances depending upon an interruption of the basal or peripheric optic pathways are infrequent.

Injuries to the head and particularly fractures of the skull not infrequently cause paralysis of the nerves at the base of the skull with visual disturbances, but in this case the sudden onset, the non-progressiveness of the paralysis and the frequency of the abducent nerve involvement will aid in the diagnosis. Finally, a very important factor in differential diagnosis is the examination of the cerebro-spinal fluid.

Ocular Symptoms of Head Injuries.[1]—Fractures of the base of the skull are the head injuries which are most apt to give eye symptoms because of the location of the optic nerves and of the central optic pathways. It is customary to distinguish between primary injuries which are the direct result of the injury to the head and secondary injuries which result from the many changes which may follow these injuries. The primary injury may affect the optic pathways in any part of their course. The secondary changes are the result of increased intracranial pressure, of areas of softening, the formation of callous, inflammatory osteitis, meningitis, encephalitis, brain abscess, pulsating exophthalmus, etc.

Primary Changes.—The injuries to the optic nerve and to the other nerves and vessels of the eye following a fracture of the base are usually indirect, though direct injuries occur particularly when the visual center in the occipital obe is involved.

It has been known for a long time that among the fractures of the base of the skull fissures and fractures passing through the **bony optic canal** are relatively frequent. The anatomic conditions are such that an injury to the bony canal must severely damage the optic nerve and usually a partial or total optic atrophy results. It is even possible that a hemorrhage may cause irreparable damage through compression of the optic nerve. The optic nerve lesion is nearly always one-sided. The ophthalmoscopic picture is negative at first, but after an interval of from two to four weeks the atrophic change in the optic nerve head with normal blood vessels appears. In some cases the immediate loss of sight following the injury was followed by an improvement in vision. This can be explained by the associated action of a hemorrhage. Rarely vision fails at some time (several days to weeks) after the injury and in these a partial improvement has been observed. The site of injury in this form of fracture is usually the forehead, and particularly the upper orbital margin; and the injury is often a comparatively slight one. Associated symptoms on the part of the brain are often absent. The defect in the field is a varying one, depending upon the form of the optic nerve injury. A concentric contraction, a quadrant or a hemiopic field defect have all been observed when the injury has caused only a partial destruction of the optic nerve. The writer has observed a case in which just half of the optic nerve was destroyed with a resulting permanent hemianopsia limited by a horizontal line. In cases which partly recover a compression of the nerve is assumed to have taken place. A bilateral loss of sight may be due to a bilateral fracture in the optic foramina or through the sella turcica.

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[1] Wagenmann, Graefe-Saemisch, Vol. IX, Part 2, p. 710.

Uhthoff, Graefe-Saemisch, Vol. XI, Part 3, p. 1453.

Boehm, Ueber Augensymptome bei Schädelverletzungen, Inaug. Diss., Breslau, 1912.

The optic nerve may be damaged by a **hemorrhage into its sheath.** In fractures of the base of the skull hemorrhages into the sheath of the optic nerve have been frequently observed. Wagenmann states that these may be due to a rupture of a vessel in the optic canal or through the escape of intracranial blood along the optic nerve sheaths. The ophthalmoscopic changes observed in hemorrhages into the optic nerve sheaths have been variously interpreted. Wagenmann states that in those cases confirmed by autopsy, slight hemorrhages in the optic nerve sheaths which occurred from fractures in the optic canal produced practically no ophthalmoscopic change. Hyperemia of the disc occurs after fractures of the skull in cases where there is no hemorrhage into the sheath. In cases in which subarachnoid hemorrhages have passed from the skull into the orbit, distinct changes due to congestion have been observed in the fundus, presenting the picture of a slight papillitis. There is no proof that hemorrhages into the sheaths will produce ischemia suggestive of a closure of the central retinal artery, and in these cases there probably is an additional factor which interferes with the arterial circulation. Hemorrhagic discoloration of the disc and of the scleral ring with subsequent pigmentation is presumably due to hemorrhages from the vessels of the retina and of the optic disc.

In the case of one-sided complete blindness from laceration of the optic nerve in the optic canal, the direct reaction of the pupil on the affected side is lost and also the consensual reaction of the healthy eye, while the pupil in the affected eye will react consensually from light thrown into the healthy eye. As in these cases the ophthalmoscopic picture at first is negative, the condition of the pupil is of great importance to confirm the complete blindness. As the consensual reaction is preserved, the pupils are of the same size in the two eyes.

A lesion of the **central optic pathways** occurring directly after an injury to the skull through blunt force may be due to a lesion of the chiasm, the optic tract, the intracerebral pathways, and the cortical visual centers. If the fracture is in the middle cranial fossa the chiasm may be lacerated, crushed or compressed. The optic tracts are exposed to the same injuries. The intracerebral pathways are less liable to injury as they are protected, though they may be injured by a fracture or by direct injury to the brain substance and by hemorrhage. Fractures in the posterior cranial fossa from a fall or a blow on the occiput are particularly liable to injure the cortical visual center in the occiput. These are usually complicated fractures with direct injury to the visual center—indirect injuries are very unusual. The central optic pathways are only injured in severe injuries to the skull, as a fracture of the sphenoid bone or of the occipital bone is only the result of a very severe injury usually associated with profuse hemorrhage and often with prolapse and escape of brain tissue. Many of the injured succumb directly to the injury and the injury to the optic pathways is not recognized. Those that escape sometimes present a defect in their sight and this may be the principal symptom.

In lesions of the central optic pathways an important clinical symptom is the hemianopic character of the visual-field disturbance. Complete blindness is observed only when the chiasm is completely destroyed or in the presence of a bilateral lesion. An injury to the chiasm has been reported in a number of cases. This has produced a bitemporal hemianopsia or blindness in one eye with temporal hemianopsia in the other eye. Binasal hemianopsia has also been observed. There are a number of reports of injuries to the tract. The intracerebral paths are affected when after injury to the side of the skull, homonymous hemianopsia is present in addition to defects in the motor or sensory tracts. Many examples of lesions of the cortical visual center have been observed. These have usually been accompanied by the presence of a complicated fracture of the skull. If the visual center on one side is injured, there is complete homonymous hemianopsia with loss of the visual halves of the opposite side. If this destruction is not complete, the visual field disturbance is incomplete. If the hemianopsia is the result of a hemorrhage it may disappear. Hallucinations and phosphenes are sometimes complained of in the blind halves of the visual field, indicating that certain parts of the visual center are still intact. The cases in which the cortical center on both sides were injured are of particular interest. If both centers are affected, a bilateral blindness results. This is followed frequently by a more or less incomplete restoration of vision on one side. In these cases after complete blindness a small area about the point of fixation is restored in one-half of the field with fairly good central vision. The association with the memory and speech centers may be interrupted. After an injury to the side of the head hemianopsia may develop as a post-traumatic late apoplexy. The ophthalmoscopic picture is negative.

To localize the site of the injury in immediate bilateral blindness after fracture of the skull, whether in the optic nerves or in the visual center, other symptoms must be looked for. If other cranial nerves are affected, a fracture at the base is probable. Injuries to the occipital bone suggest a cortical lesion. Injuries to the chiasm usually present a bitemporal defect in the field, while in a lesion further back, the hemianopsia is homonymous. The site of injury is determined from other symptoms and the pupillary reaction.

Secondary Changes in the Optic Nerve.—After-injuries to the skull embrace a number of different conditions. In one group there are cases where symptoms of congestion and papillitis are present which occur directly after injury to the skull and are caused by cerebral hemorrhage, increased intracranial pressure and hemorrhage into the optic nerve sheath. As these conditions are often transient, the changes in the optic nerve may rapidly improve. In another group of cases the optic nerve presents the picture of optic neuritis due to any one of a number of intracranial changes, particularly of an inflammatory nature. The increased intracranial pressure in these cases has often an inflammatory origin. Hematoma, large areas of softening, progressive post-traumatic hemorrhagic softening of the brain, infectious processes in the bones, in the periosteum or in the brain tissue may all be active factors. The infection may take place from the wound or from the nose, or may be of endogenous origin. In addition to serous inflammations which cause increased brain pressure and hydrocephalus, there may be purulent processes in the form of meningitis or of brain abscess. Tuberculous lesions have also followed a traumatism to the skull and syphilitic changes have occurred in those affected with syphilis. A pulsating exophthalmus after a fracture of the base of the skull will produce a choked disc. Meningitis causes a descending neuritis. In Uhthoff's series of fractures of the base of the skull, changes in the discs were present in 14 per cent. They were always bilateral and the result of meningitis, cerebral hemorrhage or hematoma of the optic nerve sheath. A rapidly developing optic neuritis or choked disc after a fracture of the base of the skull is of serious prognostic significance. In other cases the inflammatory changes at the discs were produced by other intracranial changes and complications, such as increase of pressure, meningitis, hematoma, brain abscess, etc.

It is well known that brain pressure is frequently increased after injuries to the head, and this explains the changes in the disc which occur some time after the date of the injury. In short, a loss of sight, usually one-sided with a negative ophthalmoscopic picture and with later development of simple optic atrophy, is a very much more frequent change than either optic neuritis or choked disc.

Retinal hemorrhages are not frequent. Purtscher[1] has drawn attention to the appearance of large white areas in the retina associated with retinal hemorrhages and signs of venous hyperemia at the discs after injuries to the skull. He regards these as lymph extravasations from the lymph vessels which accompany the retinal veins.

Injuries to the skull sometimes cause a condition known as traumatic neurosis which is characterized by a functional concentric contraction of the visual field and asthenopia without opthalmoscopic changes.

The involvement of the **optic nerve** can usually be readily determined by an ophthalmoscopic examination and by the functional tests. The determination of the exact cause, however, is difficult and the decision whether the condition is a post-traumatic optic neuritis or not, can often not be made.

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[1] Centralb. f. p. Augenh., Vol. XXXVII, 1913.

In addition to the optic nerve, **other ocular nerves,** particularly the **oculomotor, trochlear, abducent, facial** and **trigeminal,** are liable to injury after a fracture of the base of the skull. These nerves may be involved either singly or collectively. As a rule the site of the injury is at the base, though their centers and intracerebral paths may be involved. The most frequent ocular nerve affected is the VI, then in frequency the III, and finally the IV. The injuries of these nerves just as those of the optic nerve occur either immediately after the injury or secondarily when they are apt to be due to a late hemorrhage, meningitis, the formation of callous, pulsating exophthalmus, etc.

When the fracture passes through the roof of the orbit or the bones of the face about the orbit, the nerves to the ocular muscles may be involved in the orbit. If the lesion is situated at the apex of the orbit a number or all of the nerves may be affected. Fractures at the base of the brain frequently injure a number of the ocular nerves which are then often combined with paralysis of the facial, auditory, trigeminal and optic nerves. When the head injury is slight a fissure probably results, where the bony fragments are not displaced but a circumscribed hemorrhage takes place and the characteristic symptoms of a fracture of the base are absent, such as coma, hemorrhage from the ear, or nose, vomiting, etc. These small fissures may cause an isolated injury to a nerve through compression or laceration from the hemorrhage. The ocular nerves may also suffer after injuries where no fracture takes place but as a result of an intracranial hemorrhage or a laceration of the nerve from deformation of the skull at the time of injury. The nerves are affected secondarily by any one of the conditions previously described. Nuclear paralyses are due to small hemorrhages or other injuries in the region of the nuclei, though secondary changes are also capable of producing this paralysis. Nystagmus has been observed after injury to the skull; its exact localization is not known. The course of the ocular muscle paralysis after head injury depends on the injury. If the nerve is completely ruptured along its basal path the paralysis is a complete one and a permanent one. If the nerve is only compressed or lacerated, the paralysis may be recovered from. Hemorrhages and nuclear paralyses are capable of recovery. An orbital paralysis is always accompanied by symptoms indicating an orbital involvement; the most striking one of these is the exophthalmus. As a rule the diagnosis of an intracranial paralysis is easily made from the other symptoms of a fracture of the skull and the site is then usually at the base of the skull. The diagnosis in the cases of isolated ocular paralysis is more difficult, when the other cranial symptoms are slight. Whether the paralysis will be recovered from, can not be definitely determined at first. In general the paralysis disappears partly or completely, the prognosis usually depending upon the severity of the cranial symptoms.

The **abducent nerve** is the nerve which is most frequently involved in fractures of the skull. Panas has shown that owing to its course this nerve is most vulnerable, particularly at the place where it crosses the margin of the petrous pyramid near the apex. The other nerves, the III and IV are protected in this region by the superficial petrous sinus. The paralysis of the VI nerve occurs generally in cases where the symptoms of a fracture of the base are present, though it is also present in cases where these symptoms are slight. It is frequently an isolated paralysis, though sometimes combined with other paralyses, particularly of the facial nerve, to which a lesion of the trigeminus may be added. A lesion of these three nerves indicates a fracture of the petrous pyramid, and if the line of fracture passes through the internal auditory meatus the auditory nerve will also be involved. A combined paralysis of the three motor ocular nerves, the III, IV and VI, suggests an injury in the region of the sphenoidal fissure.

The lesion of the abducent nerve is always a basal one; a nuclear lesion is an exception. Panas has also drawn attention to the paralysis of this nerve which occurs at birth, from meningeal hemorrhage. The involvement of this nerve in pulsating exophthalmus is well known. This is explained by the close anatomic relation of the nerve to the carotid artery within the cavernous sinus. The paralysis may be primary through the injury or secondary from pressure of the distended vessel either at the base or within the superior orbital fissure. The ectatic superior ophthalmic vein may also exert pressure. Other nerves, particularly the III nerve, are involved under these conditions. Abducent paralysis is frequently a secondary manifestation in meningitis.

The **oculomotor nerve** is much less frequently involved than the abducent nerve. In statistics of 41 cases Boehm did not find a single pronounced III nerve paralysis, though isolated ptosis was found in a few cases. It is affected during its course at the base of the skull. The diagnosis of fracture of the skull is usually apparent from the other symptoms which are present. A complete one-sided ophthalmoplegia externa and interna after fracture of the base indicates a lesion of the sphenoidal fissure. This is made more probable by pain on pressure in the region of the supraorbital notch, protrusion of the eye ball, and subconjunctival ecchymosis. Curiously a basal fracture may only paralyze certain fibres and the resulting paralysis is only a partial one. In pulsating exophthalmus the oculomotor nerve is often involved; the paralysis is then frequently secondary and is recovered from after ligation of the carotid artery.

The **trochlear nerve** is the one which is injured the least frequently. It may be involved in an orbital lesion. Of particular interest are the isolated trochlear paralyses after injury to the skull. When the brain symptoms are mild a basal site of injury is likely. If it is the result of a hemorrhage or a slight laceration, recovery is possible.

Disturbance in the associated ocular movements is an unusual symptom in fractures of the base of the skull. The **pupils** do not furnish any definite date. Loss of pupillary reaction in severe injuries to the skull seem, however, to be signs of bad prognosis. When the intracranial pressure is increased the pupils at first are narrowed, the eyes are apt to be turned up and show nystagmoid movements. When the pressure is great and the stupor prolonged, the pupils sometimes dilate, particularly on the side where the greatest pressure occurs. In many cases the dilated pupil corresponds to the site of the hemorrhage, though occasionally it has been observed on the other side. It may be assumed that generally a one-sided dilated pupil in fracture of the base with symptoms of compression usually indicate an intracranial hemorrhage in the middle cranial fossa on the same side. Reflex iridoplegia has been observed after injuries to the skull.

The **V nerve** is rarely affected in fractures of the base of the skull and then usually in conjunction with other nerves. A paralysis of the V nerve, however, is particularly frequently followed by a neuroparalytic keratitis, especially when combined with a facial paralysis and lagophthalmus. In this case it is not always easy to decide whether the complicating keratitis is due to the neuroparalytic keratitis or to the keratitis e lagophthalmo. A lesion of the V nerve has sometimes been followed by herpes zoster and severe neuralgic disturbances in the distribution of this nerve.

The **facial nerve** is the most frequently involved cranial nerve in fractures of the base of the skull. Boehm found it involved in 22 per cent. of his cases. This paralysis is generally one-sided. With the traumatic facial paralysis there is a cessation of the secretion of tears on one side, resulting in one-sided crying. The facial nerve is frequently paralyzed through forceps pressure at the time of birth. Facial paralysis coming on a number of days after a fracture at the base of the skull usually indicates inflammatory changes (meningitis).

Fractures of the vault of the skull are not nearly as apt to produce eye symptoms as the ones just described. Commotio cerebri without fracture of the skull does not cause eye symptoms. A lesion of the cranial nerves under these conditions indicates a complicating fracture of the skull or hematoma. The symptoms in compression of the brain are practically those of a fracture of the skull, as these two conditions are so frequently associated, and the symptoms are produced generally by intracranial hemorrhage, increased cerebro-spinal fluid, etc.

Acute Hemorrhagic Encephalitis.—This disease usually attacks the cerebrum in children and young adults (girls) and is often the cause for cerebral infantile paralysis. It may follow any infectious disease (scarlet fever, pertussis, diphtheria, influenza, etc.), and runs the course of an acute febrile disease with cerebral symptoms. The acute inflammatory focus develops either in the central ganglia, in the cortex, or in the centrum ovale.

Bratke[1] found eye symptoms in about 40 per cent. of all cases. At Uhthoff's suggestion this author collected 100 cases with eye symptoms. These showed in about one-half of the cases disturbances of the ocular muscles, changes in the optic discs, and pupillary changes in about the same degree of frequency. The most frequent changes were disturbances of the ocular muscles in the form of nystagmus, strabismus, conjugate deviation and ocular muscle paralysis. The oculomotor nerve was the nerve most frequently involved; generally the paralysis was bilateral, though the degree varied on the two sides. Isolated ptosis as the only symptom was observed in 8 per cent. of the cases. The abducent nerve was affected somewhat less frequently. The strabismus seemed to be in some cases the

[1] Bratke, Inaug, Diss., Breslau, 1913.

result of an ocular muscle paralysis, though a concomitant strabismus was more frequent, particularly in the cases of infantile encephalitis.

The changes in the optic disc consisted in optic neuritis which was distinctly more frequent than choked disc. Optic atrophy was unusual and a number of cases are on record of complete amaurosis without ophthalmoscopic changes. The pupillary changes consisted in an absence of the light reaction and an inequality of the pupils. The loss of the light reaction and anisocoria were always prognostically a serious symptom. No deductions can be drawn from the difference in size of the pupil to the probable side on which the lesion is situated.

General Paresis.—The ocular symptoms, in order of frequency, are pupillary changes, optic atrophy, and paralyses of the ocular muscles.

Optic atrophy occurs in paresis about as frequently as in tabes, *i.e.*, in about 8 per cent. of the cases, according to Uhthoff, though from observations made in eye-clinics tabetic atrophy is very much more frequent The prognosis is always bad; cases which remain stationary or are one-sided belong to the greatest rarities, and when this does occur the diagnosis is probably incorrect. Clinically, the optic atrophy in paresis is an analogous process to that found in tabes. In paretic optic atrophy, complicating changes in the spinal cord (degeneration of the posterior columns) are always present and the cases of general paresis in which the tabetic symptoms are pronounced or precede the paralytic symptoms are more frequently complicated with optic atrophy than those with only cerebral paralytic symptoms. Uhthoff does not believe that basal neuritic processes in the optic paths and vascular lesions are the primary changes in general paresis and that the optic atrophy is secondary (Stargardt[1] and others), though these inflammatory changes are occasionally present. Proof has not been brought that the optic nerve process regularly takes its beginning from the region of the chiasm or the intracranial optic trunks, nor that it is secondary to inflammatory meningeal changes. The changes in the cortex in general paresis are believed by most authors to be primary atrophic changes in the cortical elements, particularly in the ganglion cells with secondary or simultaneous meningo-encephalitic lesions. This holds true also for the optic atrophy, in Uhthoff's opinion.

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[1] Stargardt, Ursachen d. Sehnervenschwundes bei Tabes u. Progr. Paralyse, Arch. f. Psychiatrie u. Nervenkr., Vol. LI, No. 3, 1913.

The clinical signs of the optic atrophy in paresis (course of the visual disturbance, changes in field, prognosis, ophthalmoscopic symptoms, and their relation to the visual disturbances) resemble in every particular those found in tabetic atrophy, and point to a peripheric beginning of the process, either in the retina or in the orbital optic nerve. Optic atrophy may develop in any of the various stages of paresis and sometimes precedes the true symptoms of general paresis. The latter is especially the case when tabetic symptoms precede the paralytic disturbances.

Optic neuritis, hyperemia of the disc and dilatation of the retinal vessels are uncommon findings in general paresis; they are probably due to a complicating syphilitic basal meningitis, arteriosclerosis, or hydrocephalus. A partial atrophic discoloration of the discs in their temporal halves with diminished sight is also due to a complicating lesion. Some authors have described hyperemia of the disc and of the retina with slight clouding. Uhthoff found this in only 2 per cent. of his cases, and regards these changes as physiological. Klein[1] has described a retinitis paralytica which he found in general paresis in 62 per cent. of the cases and in other mental conditions. This retinitis is said to have the two following characteristics: (1) a diffuse light clouding of the retina over the fundus without inflammatory symptoms; (2) a peculiar change of the vessel walls, particularly of the arteries, consisting in dilatation for a short part of their course. In some cases only one of these symptoms is present, though in the majority both are supposed to exist. Uhthoff found that the diffuse opacity in the retina was also present in other mental diseases and even in health, and does not regard the vascular changes at all characteristic for paresis. In a small percentage of cases of severe alcoholism there is a slight clouding of the disc and of the surrounding retina, particularly in beginning toxic amblyopia which is pathological.

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[1] Klein, Wiener med. Presse, No. 3, 1877.

The changes in the **visual field** of paretics suggest a peripheric beginning of the atrophic process. Hemianopsia is very uncommon, and the bitemporal variety is not observed. Uhthoff found bitemporal hemianopsia to be absent in cases of paresis and of tabes, as well as homonymous hemianopsia, which speaks against the beginning of the atrophic optic-nerve process in the region of the chiasm and of the optic tracts as Stargardt has recently suggested.

Paralysis of the **ocular muscles** occurs in about 10 per cent. of the cases; this would be about one-half as frequent as in tabes. Just as in tabes the oculomotor nerve is the one most frequently affected of the motor ocular nerves. Ophthalmoplegia externa and totalis are not infrequent in paresis and may precede the paralytic symptoms. In fact chronic progressive ophthalmoplegia is rarely isolated and is usually complicated with other diseases of the central nervous system, particularly tabes, paresis or a combination of these two. The paralysis of the ocular muscles in paresis is analogous to the tabetic paralysis; and is especially frequent in the cases of tabo-paresis. Isolated internal ophthalmoplegia is present in 3 per cent. of the cases, as compared to 5 per cent. in tabes (Uhthoff).

The **pupillary changes** in paresis have great diagnostic significance. Reflex iridoplegia was present in 44 per cent. of Uhthoff's cases, in 9 per cent. of these it was associated with loss of accommodative reaction—in other words, the paralysis was complete. In an additional 8 per cent. the reaction to light was very slight and in 16 per cent. the light reaction was sluggish but distinctly pathological in the presence of a prompt convergence reaction. This makes a pupillary disturbance in 68 per cent., which is considerably less than in tabes. The reflex iridoplegia is not infrequently one-sided or is more pronounced on one side than on the other. The one-sided pupillary changes have the same significance as the bilateral ones. Total iridoplegia is a symptom which is decidedly more frequent in paresis than in tabes; some authors have found it present in 16 per cent. Difference in size of the pupil is a frequent symptom in general paresis; according to the statistics of various authors, this difference in the pupils is present in 64 per cent. of the cases (Uhthoff).

Dilatation of the pupil to sensory stimuli is often lost together with the light reaction. Holden[1] found that the absence of the sensory reflex of the pupil was a constant early symptom in paresis. Irregularity of the pupillary margin is not an infrequent symptom, though not characteristic for paresis and it also occurs in other diseases, such as tabes, cerebral lues, etc.

Signs of old intraocular disease suggestive of a previous syphilitic process are very infrequent.

Differential Diagnosis.—The optic atrophy and the ocular muscle changes are slightly less frequent in paresis than in tabes, and this holds even more for the pupillary changes. Spinal miosis is more frequent in tabes than in paresis, while differences in the size of the pupils, irregularity and total iridoplegia are more frequent in paresis. In cerebral lues the lesion of the optic nerves, of the optic muscles and of the pupils are of a different character than in either of these conditions. Simple progressive optic atrophy does not occur in cerebral lues. Hemianopic disturbances however are much more frequent in this disease. The ocular muscle paralyses are also more frequent and have the characteristics of basal and peripheric paralyses. Pupillary changes are not as frequent in brain syphilis as in the other two conditions and are usually the remnants of old oculomotor pareses. It is very unusual to have a reflex iridoplegia of syphilitic origin remain an isolated and uncomplicated symptom, while pronounced anisocoria with preserved pupillary reaction and one-sided ophthalmoplegia interna are very much more frequently isolated symptoms and do not necessarily indicate a future complication with tabes or paresis.

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[1] Holden, J. Nerv. and Ment. Dis., XXXII, 1904.

Idiocy.—In this affection the congenital or early onset of mental deterioration is characteristic. Hereditary psychopathic tendencies, syphilis and alcoholism of the parents are of etiological importance.

Uhthoff[1] finds from a study of the literature that hypermetropia was present in about half the cases. The frequent presence of this refractive error is significant though it is only an indication of the arrest of development of the eye just as a similar defect exists in the brain. Muschallik,[2] at Uhthoff's suggestion, collected 100 cases of idiocy with ocular symptoms. The ocular symptoms in the order of frequency were strabismus, nystagmus, optic atrophy and pupillary disturbances.

Strabismus was found in 38 per cent. of these cases, usually in the form of convergent strabismus. In about one-half of these cases the condition was associated with optic atrophy. Distinct ocular paralysis was not frequent (5 per cent.) and the cerebral changes that are found in idiocy do not usually lead to paralysis of the ocular muscles. Nystagmus is a more frequent symptom (28 per cent.) and pathological changes were found in the brain in the cases where an autopsy was performed. The nystagmus was frequently complicated with other changes in the eye, such as coloboma of iris and choroid, optic atrophy, zonular cataract and strabismus. Disturbances in the pupillary reaction were comparatively infrequent. Atrophy of the optic nerve was found in 25 per cent. of the cases. The ophthalmoscopic picture was that of a simple atrophy, though in a part of these cases the atrophy must have been neuritic. Certain congenital anomalies of the eye, cataract, microphthalmus, etc., are more frequently found in idiocy than in normal people. This is easily explained by the association of idiocy with congenital developmental disturbances of the brain.

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[1] Uhthoff, l. c., p. 1555.

[2] Augenerscheinungen bei Idiotie, Inaug. Diss., Breslau, 1913.

A. W. Ormond[1] examined 43 Mongolian imbeciles and found the following eye changes: Blepharitis, conjunctivitis, the interpalpebral fissure is directed up and out. In over 50 per cent. there were changes in the lens, the cataract is incomplete and in the form of a single opaque mass in one meridian of the lens or a completely formed lamellar cataract. A star-shaped opacity may be present at either the anterior or posterior pole, which is probably a later development. The teeth do not show the honeycombed condition seen in lamellar cataract, but the skin and the hair exhibit characteristic changes.

Amaurotic Family Idiocy.—This disease is one of a severe, fatal cerebral lesion with characteristic eye changes occurring in children. The eye changes were described by Waren Tay,[2] though the first complete description of the disease was given by B. Sachs[3] of New York.

The ophthalmoscope in these cases reveals a picture somewhat resembling that of closure of the central retinal artery. There is a round white area in the region of the macula lutea with a brownish-red spot in the center. At a later stage the optic nerve becomes atrophic, though the changes in the macula lutea persist. The cases have all occurred in children under one year. A number of members of the same family are affected and all the cases have died before the end of the second year. A remarkable feature is that the condition has only been observed in Jewish children. The children at birth and during the first few months appear perfectly normal, then become apathetic and apparently lose their sight. They are not able to hold up their heads nor sit up. The extremities become weak and the picture of a more or less complete diplegia develops. Convulsions have been observed. The reflexes are sometimes increased. Mental development of the children is arrested at an early stage and the children become idiots and blind. Gradually marasmus sets in and the patients die as a rule before the end of the second year.

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[1] T. O. S., 1912.

[2] Waren Tay, T. O. S., Vol. I, p. 55, 1881; Vol. IV, p. 158, 1884.

[3] B. Sachs, Journal of Nervous and Mental Diseases, 1887, 1892.

Sachs has described the following characteristics: (1) psychical defect noted in the early months, leading to complete idiocy; (2) a weakness of all the extremities, progressing to complete paralysis; (3) reduction of vision which leads to complete blindness, characteristic changes in the macula lutea and subsequent optic atrophy; (4) marasmus and fatal termination before the end of the second year; (5) the disease is apt to affect a number of children in the same family. Other occasional symptoms are nystagmus, strabismus, hyperacousis or loss of hearing. Though the changes in the macula are striking and an important symptom, the disease can be diagnosticated in the absence of this symptom, according to Sachs, and the cerebral symptoms have been known to be developed some months before the retinal changes. Though the disease usually terminates before the end of the second year, Sachs has observed the case of one child which attained the age of five and one-half years; in this case the characteristic symptoms were present and two or three members of the same family have had the same symptoms. Koller[1] reports that the macular changes may occur after the optic atrophy.

Pathological examinations have shown a disease of the cerebral cortex and an involvement of the pyramidal tracts. A number of eyes have been examined and have shown a degeneration of the ganglion cells of the retina similar to that found in the ganglion cells of the brain.

[1] Koller, N. Y. Medical Record, 1896.

The nerve fibres of the optic nerves and tracts were also affected, probably secondary to the retinal changes. There is some doubt as to the existence of an œdema in the region of the macula. An explanation of the remarkable ophthalmoscopic picture has not been given.

Dementia Præcox.—Though distinct pathological changes in the eye grounds have been described in dementia præcox, these findings have not been confirmed by other authors and Uhthoff[1] and Albrand[2] regard them as most improbable. On the other hand, this disease possesses definite pupillary changes.

The most striking symptom in dementia præcox is the pupillary reaction. The light reaction of the pupil is not lost but there is a loss of the physiological pupillary unrest as well as of the dilatation on psychical and sensory reflexes. This has been particularly studied by Bumke,[3] who found these pupillary phenomena to be absent in 60 per cent. of the cases. In the determination of this symptom the method of examination is important. Thus, Runge[4] found that in the examination with a source of light of nine-meter candle strength the result was more constant than with daylight. The examination should be carried on in a dark room with a definite artificial source of illumination and by means of the loupe. The eye to be examined must be quiet and the accommodation must not vary. This symptom is of great diagnostic importance in the field of paranoia and maniacal depressive insanity. If in these cases the pupillary unrest and the dilating reflexes are absent, or if they are reduced when organic disease can be excluded, the cases belong to the catatonia group.

In dementia præcox Westphal's catatonic iridoplegia is of importance. This is not the typical reflex iridoplegia to light, but is a complete iridoplegia to light as well as on convergence. It is an unusual and transient symptom and occurs only in the cases with severe and protracted periods of stupor, where the pupils are usually widely dilated and there is a change in the outline of the pupil. The size of the pupils in dementia is usually larger than normal.

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[1] Graefe-Saemisch, p. 1560, Vol. XI.

[2] Albrand, Arch. f. Augenheilk., Vol. LXVI, pp. 114 and 229, 1910.

[3] Quoted from Uhthoff.

[4] Qouted from Uhthoff.

CORPORA QUADRIGEMINA AND PINEAL BODY

Though the symptoms of lesions of the quadrigeminal bodies are difficult to isolate from those of the surrounding parts, affections of this region, in which must be included the pineal gland, often give definite eye symptoms. Uhthoff collected 88 cases which were controlled by autopsy and which exhibited eye symptoms; in 80 per cent. of these the lesion was a tumor (tubercle, gliosarcoma, etc.).

Ophthalmoscopic changes are very frequent, in about 75 per cent. of the cases. These consisted in **choked disc** and optic neuritis which are easily explained by the proximity of the quadrigeminal bodies to the aqueduct of Sylvius; any pressure exerted by a tumor in this region will cause an internal hydrocephalus. There are no characteristic disturbances of the visual field.

A nearly constant symptom is a defect in the ocular muscles, particularly in those supplied by the III nerve, and in many of the cases both III nerves were affected. A characteristic and distinctive symptom of a lesion in this region is a disturbance of **associated ocular movements up or down,** without any other ocular-muscle defect. This was present in about 19 per cent. of Uhthoff's cases and generally in both upward and downward directions. The associated lateral movements are not involved. Of the bilateral III nerve disturbances, ptosis is the most frequent and is often the only symptom. The IV nerve is involved very much less frequently, as it is situated at some distance from the quadrigeminal region. The VI nerve is affected still less frequently.

Disturbances of the **pupillary reaction** are present in one-half of the cases. This is easily explained by the frequent involvement of the oculomotor nerve and of the optic nerves. **Nystagmus** was present in 12 per cent. of Uhthoff's cases; this is more frequent than in any other intracranial disease. Nystagmus was associated with vertigo, disturbance of equilibrium, cerebellar ataxia, etc. In about 25 per cent. of the cases there was a disturbance of the **auditory nerve.** Loss of hearing with ocular symptoms (particularly of the III nerve) are important for diagnosis of lesions in this region. The VII nerve is affected in the same frequency.

Characteristic symptoms are choked disc, loss of vertical associated ocular movements, disturbed pupillary reaction, nystagmus and involvement of the VII and VIII nerves.

CEREBRAL PEDUNCLES

In about one-half of the lesions involving the peduncles tumors are present, and in these tuberculosis in the form of a solitary tubercle preponderates. This is followed in frequency by softening and hemorrhage. Motor and sensory disturbances of the opposite half of the body are regularly present, usually complicated with a lesion of the III nerve on the same side. The III nerve paralysis is the only eye symptom. A hemiplegia without oculomotor involvement is infrequent and but few cases have been observed of involvement of the oculomotor nerve without hemiplegia. The most frequent and characteristic symptom of a peduncular lesion is the **alternating oculomotor** and **body paralysis,** hemiplegia alternans superior, Weber's symptom-complex. This was found in practically all of the cases controlled by autopsy. The syndrome of Benedict consists in an oculomotor paralysis with symptoms of irritation on the opposite side of the body (tremor, rigidity). It is clear that in these two groups the symptoms only differ in grade, and are produced by lesions in the same locality. When the oculomotor nerve is involved both external and internal branches are affected. Changes in the optic nerve (ophthalmoscopic changes) and in the optic paths (hemianopsia) are generally absent. Changes in the pupils depend upon the branches of the III nerve involved; a one-sided dilatation of the pupil with ophthalmoplegia interna is frequent.

THE SO-CALLED ACOUSTIC TUMORS IN THE CEREBELLO-PONTINE ANGLE

This characteristic tumor of the posterior cranial fossa, arising in the recess between the cerebellum and the pons, is usually closely related to the auditory nerve and less frequently to the facial nerve. It is encapsulated and does not invade the neighboring brain tissue. It occurs usually in people of middle age, and disturbance of hearing is probably the first and most important symptom, generally accompanied by vertigo and Ménière's attacks. There are disturbances of the extremities and in the trunk. The course is usually a slow one and lasts for from two to eight years. The **eye symptoms** are bilateral **papillœdema** and a **paralysis** of the **VI, VII** and **V nerves.**

In 90 per cent. of the cases there were changes in the optic nerves, choked disc, optic neuritis or atrophy, and peripheric contraction of the fields. The most frequent of these is bilateral choked disc.

The ocular muscles are involved in a very much smaller number of cases. The abducent nerve was affected in 25 per cent., and in half of these the involvement was a bilateral one, though an associated lateral paralysis is not a marked symptom. The III and IV nerves escape involvement. Nystagmus is quite frequent (33 per cent.). Anomalies of the pupils are absent. The facial nerve is involved in 50 per cent., the trigeminal nerve in 25 per cent. Hartmann[1] states in the differential diagnosis between these tumors and those of the neighboring parts, that when pronounced cerebellar symptoms (ataxia, vertigo, headache and vomiting) become associated with choked disc and ocular muscle paralysis, but with only slight involvement of hearing, a tumor must be suspected. Alternating severe hemiplegia with paralysis of the VI, VII, or VIII nerve, particularly with a paralysis of the associated lateral movements in the early stages of the disease, suggest a tumor of the pons. In the acoustic tumors involvement of the ocular muscles and of half of the body are unusual, while loss of hearing, particularly complete deafness on one side or bilateral deafness, is a frequent and an early symptom.

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[1] Hartmann, Zeitsch. f. Heilkunde, Vol. XXIII, p. 391, 1902. Quoted from Uhthoff.

PONS

The most frequent lesion of the pons is a tumor, and among the tumors a conglomerate tubercle comes first in frequency, second is glioma or sarcoma. The solitary tubercle occurs here just as elsewhere in adolescents; it shows a slow growth and the symptoms consequently develop gradually with the usual characteristics of compressing the nerve tissues rather than directly infiltrating them. Glioma also occurs generally in adolescents. Uhthoff found from a study of the reported cases of pontine tumor that **eye symptoms** were present in 33 per cent., consisting in papillœdema or optic neuritis, and paralyses of the VI and VII nerves.

The changes in the optic nerves were always secondary. Choked disc is frequent, and occurred in about one-third of the cases which give eye symptoms. Optic neuritis without swelling of the nerve head was found in 40 per cent. These ophthalmoscopic changes are not observed in the early cases, probably as they are the result of a secondary internal hydrocephalus.

The ocular muscles are involved in tumor of the pons in a characteristic manner. The **abducent nerve** is the one most frequently affected (80 per cent.), and in about one-half of the cases the abducent paralysis is a **bilateral** one. The close anatomic relation explains the frequent associated paralysis of the **abducent** and **facial** nerves which was observed in 75 per cent. of the cases. In these the abducent paralysis was bilateral in one-third of the cases, while the facial paralysis was present on only one side. The III nerve is usually not involved. The trigeminal nerve of the same side was affected in about one-half of the cases.

In about 50 per cent. of the cases of **abducent paralysis** there was a **hemiplegia** of the opposite side. Together with an involvement of the abducent nerve, an associated paralysis of the internal rectus of the opposite side is frequently found in tumors of the pons—in other words, a **paralysis of lateral movement.** The convergence power is preserved. A paralysis of lateral ocular movement with hemiplegia of the opposite side are characteristic symptoms of a pons lesion. A complete facial paralysis with paralysis of the lateral movements is also a typical pons symptom when there is no crossed hemiplegia.

The facial nerve is frequently involved in tumors of the pons, though perhaps not as frequently as the abducent nerve. The most frequent combination is one of facial paralysis with crossed hemiplegia (Millard-Gubler's paralysis), which suggests a lesion of the lower part of the pons. The trigeminal nerve is involved in not quite half of the cases, somewhat less frequently than either the VI or the VII nerve. It is practically never involved alone. In these cases a neuroparalytic keratitis was observed in about 25 per cent. of the cases. The pupils show nothing characteristic. Nystagmus is an unusual symptom. Other lesions of the pons like hemorrhage, softening and abscess, produce similar symptoms as a tumor, though their onset is more acute.

OPHTHALMOPLEGIA

The term, ophthalmoplegia, as suggested by Mauthner, means either a bilateral ocular-muscle paralysis, particularly if the muscles involved are innervated by different nerves, or a one-sided paralysis where a number of different nerves are affected. The term is generally used in cases where all the external muscles are more or less involved. It indicates a lesion of the nuclear portion of the ocular nerves situated in the floor of the fourth ventricle, in the aqueduct of Sylvius and in the posterior part of the third ventricle. Ophthalmoplegias which are due to peripheric or basal lesions are excluded from this description. Bilateral isolated paralysis of the abducent nerves and the bilateral isolated ptosis are not included in this description as these are frequently not nuclear in origin. Again, isolated mono- or bilateral paralysis of the inner ocular muscles is not included. In general, the condition occurs in two groups—one the acute and the subacute; the other the chronic which is often progressive in character.

Acute and Subacute Ophthalmoplegia.—These paralyses are usually due to poisoning or to infection, in a small proportion no etiological factor is ascertained.

The most frequent of the **poisons** is an acute severe form of ophthalmoplegia which depends upon alcoholism and has been called polyencephalitis acuta hemorrhagica superior by Wernicke. This is an inflammatory process in the region of the nuclei of the ocular muscles which rapidly involves the ocular nerves with the exception of those governing the interior of the eye, accompanied by severe cerebral symptoms (coma, delirium, vertigo, headache, ataxia, etc., and generally leading to death in from one to two weeks).

Another cause, though an unusual one, is chronic lead poisoning. In the cases reported, external ophthalmoplegia was associated with involvement of the inner muscles, with a disturbance of the pupillary reaction as well as of the accommodation. The course is usually a subacute one and a frequent complication is optic neuritis. Severe cerebral symptoms are absent. Other symptoms of lead poisoning are always present, such as headache, convulsions, hemiplegia and vertigo.

A more frequent cause is botulismus (poisoning from meat, sausage, fish, oysters, etc.). In all cases there was a more or less complete bilateral internal ophthalmoplegia with dilated and immobile pupils. In about one-half of the cases there is bilateral ptosis and some of the external ocular muscles are also involved. A number of cases have been reported where the ophthalmoplegia was complete. A characteristic is that the inner ocular muscles are particularly prone to involvement. This has been demonstrated by experimentation on monkeys. In the recent investigations of Römer and Stein[1] subcutaneous injection of botulismus-toxin in monkeys produced dilated pupils and at autopsy the ganglion cells in the ocular motor nuclei were found degenerated. The general symptoms of poisoning (vertigo, headache, prostration, difficulty in swallowing, dryness of throat, diarrhœa or constipation, distention of the stomach, difficulty in urination) may occur before the onset of the ocular symptoms or simultaneously with them. The eye symptoms were observed at a time after the poisoning varying from a few hours to ten or fourteen days. The characteristics are the regular involvement of the inner ocular muscles, the frequent ptosis, the bilateral appearance, the absence of fundus changes, the character of the complicating general symptoms and the recovery.

Paralyses of the ocular muscles have been reported after coal-gas poisoning.

The most frequent **infections** which give rise to ophthalmoplegia are diphtheria and influenza. All cases of ocular-muscle paralysis after diphtheria are of course not included under this heading, particularly the many cases of bilateral paralysis of the accommodation. A number of cases of bilateral multiple ocular-muscle paralysis have been reported in which the symptoms of pronounced external ophthalmoplegia were present. The pupillary reaction to light was preserved, while the accommodation was paralyzed in both eyes. The patients usually show general motor weakness, absence of knee-jerk, etc. Uhthoff has only observed one case. One-sided or bilateral paralysis of the abducent nerve after diphtheria is not regarded as an ophthalmoplegia, though it probably is a nuclear lesion. The cases of VI nerve paralysis are quite frequent and occur in about from 6 to 12 per cent. of the cases of post-diphtheritic ocular paralysis.

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[1] Graefe's Archiv, Vol. LVIII, 1904.

Ocular-muscle paralysis after influenza occurs in about 8 per cent., according to Groenouw, though a more or less complete ophthalmoplegia is very unusual. Cases of one-sided ophthalmoplegia after influenza have been reported in which the external muscles or the internal muscles have been involved. In the bilateral cases the process is probably a polyencephalitis. At the same time, peripheric changes such as occur in polyneuritis may also explain these ocular-muscle paralyses. External ophthalmoplegia has also been reported after measles, typhoid fever, cerebrospinal meningitis, pneumonia, whooping cough and acute rheumatism.

Syphilis is a very infrequent cause for the acute or subacute nuclear ophthalmoplegia. Ophthalmoplegia has been reported after trauma, resulting from hemorrhages and softening in the region of the nuclei of the ocular nerves, though traumatic ocular paralyses are much more frequently peripheral or basal. In a peripheral and basal paralysis, there are evidences of fracture of the skull and involvement of other basal nerves. The presence of diabetes and the symmetrical occurrence of the ocular paralysis, suggest a nuclear lesion. This does not hold for the bilateral paralysis of the abducent nerve which is frequently basal. A complete and permanent paralysis is more suggestive of a peripheral and basal origin. In a number of cases of ophthalmoplegia no cause could be determined.

Chronic Progressive Ophthalmoplegia.—A. This occurs without other nervous symptoms, and Uhthoff found it in 14 per cent. of the cases. In one-third of the cases the affection begins in childhood up to the sixth year; the second and third decade are frequently involved, while later in life it is very unusual. An ophthalmoplegia which began in the earliest years may later in life show signs of progression. As a rule the tendency to progress is present, though there may be distinct intermissions with improvements. The lesion is always bilateral, though not necessarily symmetrical.

Diplopia is complained of in only a small proportion of the cases. The internal ocular muscles are always intact. Ptosis is of moderate degree, though it is regularly present and is often the first symptom. The degree of the paralysis varies and is frequently more pronounced in the morning than toward evening; it is regularly made worse on exertion, excitement or fatigue. The condition generally leads to a complete paralysis of all the external ocular muscles. The process may remain localized to the ocular symptoms or after a series of years other complications (tabetic symptoms, etc.) supervene. Ophthalmoscopic changes are not present. If they are present, they always indicate complications on the part of the brain (tumor, tabes, etc.). In addition to the external ophthalmoplegia, the ocular facial nerve is sometimes involved. Syphilis is not the cause for this condition when uncomplicated.

B. *Chronic Progressive Ophthalmoplegia Complicated with Tabes and Paresis.*—The form of chronic ophthalmoplegia which is complicated with other diseases of the nervous system is more frequent than the above-described uncomplicated isolated form. The complications in order of frequency are: tabes, paresis, tabo-paresis and occasionally psychic disturbances. These complications are present in practically half of the cases, and in three-fourths of these the complication is tabes. In 14 per cent., according to Uhthoff, in addition to the tabes, there are symptoms of paresis, while in only 7 per cent. paresis alone is the complicating nervous affection. This shows the great diagnostic importance which progressive chronic ophthalmoplegia has for tabes, and paresis.

Ocular-muscle paralyses in 20 per cent. of the cases occur in either tabes or paresis. The percentage of this origin for chronic progressive ophthalmoplegia is, of course, very much higher. Ophthalmoplegia in tabes or paresis, like the usual ocular-muscle paralysis, may precede the other condition for many years. The ophthalmoscopic changes consist in an optic atrophy (30 per cent. of the cases, Uhthoff) so that the combination of optic atrophy with external or total ophthalmoplegia points with great probability to tabes or beginning paresis, even in the absence of other symptoms. Disturbances of the pupil and of the inner ocular muscles are extremely frequent. Thus a reflex iridoplegia to light or total, in part with anisocoria was found in more than half of the cases, and a paresis of the inner ocular muscles was present in 25 per cent., so that in 78 per cent. of the cases changes in the pupil were found present. Pupillary disturbances, in addition to the optic atrophy, in ophthalmoplegia is of still greater importance in the diagnosis of either tabes or paresis. The facial and the trigeminal nerves were found affected in only a small proportion of cases.

Chronic progressive ophthalmoplegia has been observed in combined disease of the posterior and lateral columns, in multiple sclerosis, syringomyelia and paralysis agitans. A paresis of individual ocular muscles is not an infrequent symptom in exophthalmic goitre; a true ophthalmoplegia is however very unusual. Ophthalmoplegia has also been described in diabetes.

Congenital nuclear ophthalmoplegia shows multiple congenital defects, usually with ptosis and intact inner ocular muscles. In more than half of these cases in addition to ptosis there was a complete external ophthalmoplegia. In other cases the disturbances were only partial. The inner ocular muscles were always uninvolved. In some of these cases certain hereditary factors were noticeable, inasmuch as a number of members of a family presented similar disturbances and other congenital anomalies of the eyes were also found present.

MYASTHENIA GRAVIS*

The disease usually begins insidiously and runs an intermittently progressive course. Though it can affect every muscle, it is localized particularly in the muscles supplied by the cranial nerves and in the majority of moderately advanced cases the typical picture of ptosis and weakness of the facial nerve is present. As the patient begins to speak, the nasal speech is striking. The symptoms develop in a descending line, after a disturbance of the ocular muscles, a weakness is observed in the muscles which govern mastication, the face, speech and swallowing. Finally, the muscles of the neck, of the back and of the extremities are affected. This sequence may be reversed or be irregular in the onset of the symptoms. At the acme of the disease the process is always an extensive one and may involve all the voluntary muscles of the body, particularly those of the bulbar nerves.

The characteristic sign in the affected muscles is their abnormally rapid fatigue (myasthenia). At the beginning the muscles respond to the nerve impulse, then as this impulse is repeated they gradually become weakened and simulate a paralysis; while the contraction steadily becomes less energetic. Thus the changes are always less marked in the morning than they are later in the day. In the final stages of the disease true paralytic conditions are present without atrophic disturbance of the muscles or a reaction of degeneration. With the abnormal rapid fatigue the muscles show a similar fatigue on electric stimulation,

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*Uhthoff, l. c., p. 459.
Lewandowsky, Lewandowsky Handbuch d. Neurologie.

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the so-called myasthenic reaction discovered by Jolly. The intensity of the symptoms varies and a marked improvement sometimes takes place.

Oppenheim[1] describes this disease as consisting in a combination of bulbar symptoms with an incomplete external ophthalmoplegia and weakness of the muscles of the back and of the extremities which usually develop slowly. The muscle of mastication is particularly involved, and a paresis of the orbicularis, a frequent involvement of the muscles of the neck, and the preponderance of myasthenia over true paralysis are all characteristic features. Furthermore, the intermittent course, the absence of true muscular atrophy and the presence of a myasthenic reaction, are also important symptoms.

The commencement is often, as has been stated, in the eye muscles of which ptosis is the most frequent symptom and is practically present in all cases. It is always bilateral, though one eye may be affected later than the other. The signs of fatigue are particularly noticeable in the ptosis. Any continuous attempt to fix, to raise the lids, to speak, bodily exertion, etc., are factors which increase the ptosis. In the evening the upper eye lids are practically closed. Most of these cases are complicated with other disturbances of ocular motility in the form of a more or less marked external ophthalmoplegia. In some of the cases the disease remains localized to the eye muscles (Karplus' asthenic ophthalmoplegia) and in many cases are referred to the neurologist by the oculist with a diagnosis of ocular paralysis.[2] In the cases of pure fatigue the patients awake in the morning appearing quite normal and without diplopia. Then in the course of a number of hours, the symptoms of paralysis gradually set in. The extent of the paralysis varies up to the point of the complete external ophthalmoplegia, and if at all pronounced, is usually bilateral. The disturbances in the ocular muscles show a distinct myasthenic character, though they may at times seem to be true paralysis.

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[1] Lehrbuch d. Nervenhilk., p. 1365, 1913.

[2] Lewandowsky, l. c.

An involvement of all the outer ocular muscles has been reported in a number of cases, while in other cases the involvement of these muscles was an incomplete one. The function of the internal recti in convergence has also been found disturbed in a characteristic way by some authors. The internal ocular muscles, ophthalmoplegia interna, were never found affected. The facial nerve was involved in 50 per cent. of Uhthoff's cases. The weakness of the orbicularis is also always bilateral, of a myasthenic character, and in many of the cases is an isolated involvement of the upper division of the facial nerve. The pupils show no noteworthy disturbances. Pathological fundus changes have not been observed in any of these cases, though symptoms of fatigue and a functional disturbance of the optic nerve and of the retina (visual field) have been described.

The pathology of this disease is not understood; it is probably an affection of the nuclear region of these nerves, because of the grouping and the form of development of the symptoms, as the muscles, particularly those of the eyes, are involved according to the anatomic relation of their nuclear origin. Furthermore, the ocular-muscle paralyses resemble those found in nuclear ophthalmoplegias. Some authors believe that in a part of the cases the disease depends upon a muscular origin. The differential diagnosis may sometimes be difficult, though associated disturbances of other muscles of the body and particularly the presence of the myasthenic symptoms with absence of the reaction of degeneration are important.

The functional disturbances found in hysteria are usually different. The symptoms of fatigue are not the same and the rapid fatigue of the muscles on repeated innervation and on electric stimulation with a good function at the beginning and a rapid restitution of the function after a rest, are all characteristic of myasthenia. Other general nervous symptoms are apt to be absent in myasthenia.

Ocular paralyses and bulbar symptoms do not belong to the picture of functional nerve disease. Thus hysterical ptosis is not a true ptosis, but a pseudo-ptosis caused by an orbicularis spasm and in hysteria a paresis of the orbicularis muscles and of the facial nerve are usually absent.

Medulla Oblongata.—*Bulbar paralysis* may be added to progressive muscular atrophy or amyotrophic lateral sclerosis. It sometimes precedes either of these conditions, and when in the course of tabes, multiple sclerosis or myelitis the medulla is invaded, symptoms of bulbar involvement develop. Aside from these complicating conditions, bulbar paralysis occurs as a well-defined clinical picture. In both its chronic and acute forms the absence of ocular symptoms is characteristic. In order of frequency the facial nerve is most likely to be involved, as its anatomic position readily explains. The abducent nerve comes next in frequency, while the optic pathways and the pupils are not affected. Uhthoff says that the presence of optic nerve changes in chronic bulbar paralysis always indicates a complication. The absence of eye symptoms in the acute and apoplectic form of bulbar paralysis is of diagnostic importance in localizing the lesion in the medulla.

DISEASES OF THE SPINAL CORD

Tabes Dorsalis.[1]—In tabes the pupil, the eye muscles and the optic nerve are affected.

Pupils.—Like the shooting pains in tabes, certain changes in the pupils are regarded as early symptoms. The most important change is a disturbance of the pupillary reaction. These disturbances occur in 80 to 90 per cent. and are often very early, even earlier than the lancinating pains. Oppenheim has observed cases in which an abnormal pupillary reaction has been the only symptom of tabes for ten to fifteen years. The changes consist in a loss of light reaction with active narrowing of the pupil on convergence, the "Argyll-Robertson pupil." This narrowing on convergence is even marked in miotic pupils. The reflex immobility to light is generally bilateral, though it has been observed in only one eye. A frequent preceding symptom is a sluggish contraction of the pupil. According to some authors, the pupillary immobility occasionally intermits. Iridoplegia may be complete; the convergence reaction becomes slower and then disappears. According to Knoblauch,[1] as the convergence reaction is lost the pupils gradually dilate, and with the complete iridoplegia there is pronounced mydriasis. The consensual pupillary reflex is equally affected, and the sensory reaction of the pupil is supposed to be lost in the early period of tabes. The Westphal-Piltz reflex (narrowing of the pupils on energetic closure of the lids with dilatation on exposure to light) is nevertheless found present in pupils otherwise affected.

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[1] Uhthoff, Graefe-Saemisch, II ed., Vol. IX.
Schaffer, Lewandowsky Handbuch d. Neurologie.

The Argyll-Robertson pupil is of the greatest diagnostic importance, as it occurs only in tabes, paresis, and syphilis. In tabes it is generally an early symptom and occurs in 67 per cent. of the cases. Total iridoplegia for light and convergence is much less frequent. The pupillary change rarely appears on only one side, it has then the same prognostic importance as the bilateral. Inequality of the pupillary reactions on the two sides is also not uncommon. The anatomical proof of the site of the lesion is still unknown, though it is probably situated in the reflex arc between the optic tract and the oculomotor nucleus. Absolute iridoplegia with dilated pupil and loss of accommodation occurs principally in the III nerve paralysis of cerebral syphilis.

The **miosis** is just as characteristic for tabes and is also may precede the ataxia by many years. It is not as frequent as reflex iridoplegia which is associated with it in 50 per cent. Either symptom may be monolateral. It depends upon a separate lesion, presumably in the cilio-spinal center, there producing a paresis of the dilator pupillæ.

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[1] Quoted from Schaffer.

Anisocoria occurs in 25 per cent. of the cases and is the least characteristic pupillary symptom. It is more frequent in paresis, though this symptom with preserved pupillary reaction may occur in health. If it is a symptom of organic disease, the reaction and regularity of the pupil are disturbed. **Mydriasis** is not as unusual as is generally believed, and tabes may begin with a dilatation of one pupil while the other is normally wide. Ophthalmoplegia interna occurs rarely in tabes without spinal or cerebral symptoms; it is more frequently one-sided than bilateral.

Another pupillary symptom is an irregularity of the pupil. According to Knoblauch, the loss of roundness of the pupil occurs exclusively in tabes, paresis and syphilis. Bumke states that the distorted pupil frequently becomes immobile later on, and believes that irregularities of the pupillary margin are of diagnostic and prognostic importance.

Ocular Muscles.—Important tabetic precursors are paralyses of the external ocular muscles. These appear not only in the neuralgic phase but they may be so early as to be the only existing symptom; on the other hand, they may not appear before the ataxic stage. As a rule, they are early symptoms and are usually transient. They are present for a few days and then disappear without leaving a trace. In contradistinction, the paralyses in the later period are permanent and rather tend to increase and involve other branches of the nerve. In the early period an incomplete ptosis is not infrequent, combined with pupillary changes on the same side, or a mydriasis with low or absent light reaction on the affected side, while the pupil of the other eye is medium and reacts promptly.

Paralysis of the ocular muscles occurs in 20 to 22 per cent. of tabetics; the tabetic paralysis constitutes about 20 per cent. of all muscular paralyses. The irregularity of the paralysis points to a nuclear origin and the nuclear ganglion cells have been found degenerated. The III nerve is affected most frequently, about twice as often as the VI, and seven times as often as the IV. Complete III nerve paralysis, however, is rare. Total ophthalmoplegia is unusual and generally occurs in cerebral syphilis. The levator palpebræ is often affected alone. Transient ptosis on one side or bilateral, associated with a distinct tabetic symptom, according to Uhthoff, make the diagnosis of tabes very probable. The external rectus is involved less frequently, then usually incomplete and not permanent; a superior oblique paralysis is still less frequent. Internal ophthalmoplegia and bilateral oculomotor paralysis are not frequent in tabes though quite typical for cerebral syphilis. The trigeminal and facial nerves are generally not affected. The ocular paralyses show a distinct tendency to recurrence.

Nystagmus and spasmodic convergence are very unusual symptoms. According to Curshmann, this tabetic convergence spasm is different from the hysterical one because it is isolated, while in hysteria it is combined with blepharospasm. A constant tearing is complained of by some patients.

Optic Nerve.—The early involvement of the optic nerve in the course of tabes has been known for many years. It continues unrelentingly to complete blindness. It is generally bilateral, though in the beginning it may involve only one side. The percentage of optic atrophies varies from 10 to 48, according to the statistics. Uhthoff found that 10 to 15 per cent. of tabetics suffer from optic atrophy. Every primary optic atrophy is now considered a symptom either of tabes or of paresis. The optic atrophy may be the first symptom and may precede the other symptoms by many years; it usually appears in the preataxic stage; if it does not appear then (Berger) the chances of its appearing at all are very much less. Cases with optic atrophy seem to run a benign course in reference to ataxic symptoms; Gowers has observed a case where twenty years elapsed before the ataxia came on.

In the ophthalmoscopic picture the outlines of the disc are sharply defined, the color is white with a tinge of gray or blue; generally this change precedes the visual disturbance. The retinal vessels show but little change, though they later become somewhat contracted. According to Gowers, the disc becomes gray, spotted and opaque, with sharp margins. von Grosz[1] describes two stages: in the first the disc is gray, the vessels unchanged, there is no prominence and there is a dull gray discoloration over every part of the disc. After some years the disc became whiter, particularly in the external half, and the openings in the lamina appear. v. Grosz believes the first stage to be a degeneration, while in the second a destruction of the nervous elements takes place. Leri[2] also distinguishes two phases, the first a grayish discoloration of the disc lasting for only a short time, up to one or two years, while the second stage is characterized with the white disc and an increasing loss of sight.

The loss of sight is gradual, insidious, generally affecting one eye before the other, and leads on an average to blindness in two to three years.

Uhthoff distinguishes two types of **field disturbance:** I. The optic nerve is uniformly involved. Loss of central vision with peripheric field contraction, the color field for green and red are first affected. II. Partial involvement of the optic nerve. The affected part is a sharply defined sector or a peripheric contraction with preserved central function. The subgroup where the visual disturbance begins as a central scotoma is an unusual one. This represents but 2 per cent. in Uhthoff's experience and in practically all of these there was some peripheric disturbance present. This holds also for the hemianopsias. The tabetic optic atrophy is a disease starting from the periphery and ascending from the retina. Disturbance of color sense is often an early and characteristic sign. In the first type even with nearly normal vision the color fields are affected.

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[1] Grosz, Die Erscheinungen d. Tabes am Auge, Zeitschrift f. Augenheilk., Vol. II, p. 503, 1899.

[2] Leri, Etude du nerf optique dans l'amaurose tabétique, Nouv. iconogr. de la Salp., 17, No. 5.

According to Sänger,[1] there are three forms of visual-field disturbance: (1) Loss of central vision and irregular peripheric defect. The color fields gradually diminish, loss of green, then red, yellow, and finally blue. The optic nerve is involved in its entire diameter. These cases run a rapid course. Pathologically a simple degeneration without inflammation; mercury is contraindicated. (2) The optic nerve is partly attacked by the atrophy. The defect in the field is sharply separated from the still normal part. This rarely remains stationary. The fields show sharply defined sector-like defects which include one or more sectors. (3) Uniform concentric defect with good central vision and good color perception in the preserved parts. In these cases there is a ring-form atrophy. In this group it is difficult to determine whether a perineuritic optic neuritis is not present like in syphilis.

Fuchs[2] does not believe that any deduction as to prognosis can be drawn from the fields. In addition to the ordinary cases, there are three very unusual types of field defect: (1) Marked concentric contraction with good central vision, like glaucoma. (2) Central scotoma. This scotoma is at first round, central and relative. It enlarges to include the blind spot and becomes absolute. With this there is an accompanying peripheric contraction and the eye becomes blind. This distinguishes it from the usual toxic form; the optic nerve is, moreover, pale from the beginning. (3) Bitemporal hemianopsia. This results from a tabetic focus in the chiasm; on the other hand, a complicating syphilitic pachymeningitis at the chiasm may complicate tabes.

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[1] Sänger, Ueber die Tabische Sehnervenatrophie. Verh. d. Ges. deutscher Nervenärzte, 1908.

[2] Fuchs, Tabes und Auge W. kl. W., 1912, No. 14.

According to Sänger, hemianopic defects indicate cerebral lues. A central scotoma may be present in acquired as well as in hereditary lues, as a result of a neuritis of the papillo-macular bundle; this does not occur in straight tabes. A sharply circumscribed central scotoma must not be confounded with a diminution in color perception in the central field as occurs not infrequently in tabes. Examination of the eye furnishes the best means to distinguish between tabes and pseudo-tabes luetica or to recognize a combination of tabes and cerebro-spinal lues. Cerebrospinal lues and tabes are often very much alike; they may be continuous, thus the optic nerve of a tabetic may show neuritic changes (tabes with syphilitic meningitis). As for tabes and pseudo-tabes leutica, the atrophic process in the optic nerve in the latter condition is always secondary, descending from a central inflammatory affection; it is purely a degeneration. In true tabes the optic atrophy is always bilateral. One-sided optic atrophy speaks for syphilis and variations in vision characteristic for syphilis can be observed combined with varying functional disturbances in the other cranial nerves. Furthermore, in tabes loss of vision always shows ophthalmoscopic changes, while in cerebral lues the fundus may be normal and the pallor of disc develop later.

The syphilitic infection preceding tabes is usually a mild one. It has repeatedly been observed that the usual manifestations of syphilitic eye disease are absent in those afflicted with tabetic atrophy, furthermore the latter patients seem usually strong and healthy.

Juvenile Tabes.—Cantonnet[1] concludes from 89 cases of juvenile tabes that this rare disease is always due to hereditary syphilis or syphilis acquired in earliest youth. Direct tabetic heredity is rare. As opposed to the tabes of adults, the juvenile form usually affects girls (two-thirds of the cases). The first symptom is apt to be disturbances of the bladder and amblyopia. Ataxia is about

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[1] Cantonnet, Archiv d'ophthal., 1907, p. 708.

50 per cent. less frequent than in the tabes of adults. Arthropathies are more frequent. Absolute iridoplegia is rare. Ocular paralyses do not occur as frequently as in adults. In one case monocular diplopia was described and nystagmus. In about 14 per cent. of the cases other symptoms of hereditary syphilis were present in the eyes; keratitis, chorio-retinitis. Optic atrophy is very frequent, 43.9 per cent. The prognosis for sight is worse, while for life it is better than in adults. Juvenile tabes may go into paresis.

According to Barkan,[1] primary optic atrophy occurring in children with reflex iridoplegia, anisocoria, loss of knee and ankle reflexes, no ataxia, lancinating pains, paræsthesias, trophic and bladder disturbances, are cases of juvenile tabes on an hereditary or acquired base, and resemble adult tabes. Optic atrophy is relatively more frequent, the Romberg sign and the ataxia are less frequent. Prognosis is bad for sight. Barkan has been able to collect 16 additional cases to the 51 mentioned by Marburg.[2]

Stargardt,[3] in his very important monograph on the causes of optic atrophy in tabes and progressive paralysis, states that the belief that optic atrophy in tabes and progressive paralysis is due to an ascending degeneration of the nerve and that it begins in the ganglion cells of the retina and that it is to be regarded as a system disease, is an unproven hypothesis. His examination of well-preserved retinas have never shown changes which are to be regarded as primary. The peripheric part of the orbital optic nerve has never shown changes which can be regarded as primary. The development of the optic atrophy, according to this author, takes place only in the intracranial part of the optic nerve, in the chiasm, and very infrequently in the posterior part of the orbital optic nerve, in the tract and in this geniculate bodies. In these places he has shown that not only degenerative but also exudative processes can be observed, the exudative processes preceding the degenerative ones. The exudative processes resemble the inflammatory changes of the brain cortex in paresis, both as regards their cellular elements and their vascular changes. Both processes, exudative and degenerative, depend upon the same infectious agent, which can only be regarded as the spirochete.

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[1] H. Barkan, Zur Frage der Infantilen und Juvenilen Tabes, Wiener klin. Wochenschr., 1913, No. 11.

[2] Marburg, W. k. W., 1908, No. 13.

[3] Stargardt, Arch. f. Psychiatrie u. Nervenkrankh., Vol. LI, 3.

Degenerative and exudative processes may be observed in the tissue surrounding the chiasm. They may also attack the olfactory nerves, the oculomotor nerves, the central gray and the adjoining brain, even the hypophysis. These inflammatory processes the author believes belong to the group of the late syphilitic non-gummatous diseases which include aortitis, choroiditis, arthropathies.

These views, though presenting many new and suggestive ideas, have not been accepted, principally because they do not conform to certain clinical features.

The ocular symptoms of other system diseases of the spinal cord are of but little significance when compared to their importance in tabes.

Hereditary Ataxia (Friedreich's Disease).—This condition occurs in a number of members in the same family, always before the twentieth year, usually between the fifth and the fifteenth. The principal symptom is ataxia of the lower extremities. The strength of the muscles is preserved, sensation is not disturbed, there are no cerebral symptoms except disturbances of speech and nystagmus.

The only and constant eye symptom is *nystagmus*, which is not the usual oscillating nystagmus but consists in nystagmic movements, particularly in the lateral excursions of the eyes similar to the form observed in multiple sclerosis. Many authors regard nystagmus as an ataxia (motor incoördination) of the ocular muscles.

Posterolateral Sclerosis.—The ocular symptoms are the same as found in tabes, though they are not nearly as frequent as in the latter condition. Their frequency depends upon the degree to which the posterior columns are involved. The symptoms consist in progressive optic atrophy, iridoplegia and paralysis of the ocular muscles, secondarily an involvement of the trigeminal and facial nerves.

Congenital Spastic Paralysis (Little's Disease).—This corresponds to spastic spinal paralysis with the addition of cerebral complications. These cerebral changes are often the primary ones and the motor tracts in the cord are secondarily affected. Premature birth, difficult labor, compression of the skull during labor, are all of etiological importance.

An important ocular symptom is *strabismus* which is usually concomitant, rarely paralytic. This strabismus does not differ particularly from the usual concomitant squint, though it is said to be more frequently alternating. Divergent strabismus is much more unusual. The strabismus is a cerebral symptom whose origin is not understood. The symptom next in importance is *nystagmus*. Finally, optic atrophy has been described in a few cases.

Anterior Poliomyelitis.—As the pathological change in this disease occurs in the gray substance in the anterior horns of the spinal cord, there are no eye symptoms unless the cranial nerves are involved, which is very unusual. In the cases where ocular symptoms have been present, the clinical picture has not been that of a straight spinal infantile paralysis but there have been associated symptoms indicating involvement of the brain, of the medulla and of the meninges. If the poliomyelitic process extends upward, it will first attain the region of the facial and abducent nuclei, where it usually terminates and the oculomotor region is practically never involved. There are no records of any involvement of the optic nerves nor of pupillary disturbances. Temporary nystagmus has been observed by the writer.

Lundsgaard[1] draws attention to the occurrence of ocular paralyses in epidemics of poliomyelitis and especially in the cases which run an atypical course. Medin has shown that the cranial nerves may be exceptionally affected in poliomyelitis. In 49 children with characteristic paralysis, he found affections of the facial and abducent nerves, oculomotor paralysis in one and external ophthalmoplegia in one. In all of the cases the disease began with fever, the child then developed a paralysis of an ocular muscle. The fever may last from a few days to one week, the child complains of feeling ill, and of headache. There sometimes is stupor, pain in the extremities and pronounced perspiration, and there may be gastric and intestinal disturbances.

Acute Ascending Paralysis (Landry's Paralysis).—In this condition there is a paralysis which begins in the lower extremities and involves by traveling upwards the upper extremities and the medulla. The condition frequently terminates in death. Investigations have shown pathological changes in the peripheric nervous system. Of the ocular symptoms the ones which are most important are the **paralyses** of the ocular muscles, and of these the **abducent nerve** is the most frequently involved, generally bilaterally. This abducent paralysis may be accompanied by the involvement of other ocular muscles. Optic neuritis has been described in a few cases which, however, points to a peripheric orgin for the ascending spinal paralysis.

Myelitis.—Myelitis is an inflammatory process of the spinal cord which affects the entire cross-section at various levels or extends along the length of the cord and includes the medulla. The eyes are infrequently involved; of the ocular complications that of the optic nerves is the most important. The optic nerves are involved in the form of an optic neuritis, retrobulbar in character, which explains the marked diminution of vision with often an inconstant ophthalmoscopic picture. The loss of vision usually is sudden. It increases rapidly and often leads to blindness which, however, is only transient and is followed by more or less complete recovery. The blindness may last sometimes for a number of weeks, though in these cases recovery is still possible. In other cases the loss of vision is only partial, though this is not as frequent as the transient and complete amaurosis. Both eyes are generally not involved at the same time, and there is an intervening interval of a number of days.

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[1] Lundsgaard, K. M. f. A., p. 734, 1913.

With the loss of vision the ophthalmoscope reveals either an optic neuritis or a choked disc. A symptom which is of considerable importance is the intense pain in the orbits and in the forehead which accompanies the development of the visual disturbance. This pain is marked upon attempted movement of the eyes and upon pressure of the eye balls. The visual disturbances frequently precede the spinal symptoms by days or months. The visual fields have shown a variety of defects without suggesting a typical visual field anomaly.

Pathological examination has shown an analogy between the process in the optic nerves and in the spinal cord. The optic tracts, chiasm and optic nerves can all be involved. It is now believed that a common cause produces both processes. The cause is not known; syphilis was found present in one-third of the cases (Uhthoff) and in others there was a history of cold or of an infection. The other ocular symptoms are of secondary importance.

Multiple Sclerosis.[1]—The pathological process in multiple sclerosis is an inflammatory one with multiple foci leading to sclerosis, of insidious onset and of intermittent course. The beginning occurs probably in childhood, many years before any symptoms appear. The most important early symptoms are ocular disturbances.

The optic nerve is frequently affected (in 50 per cent. of

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[1] Uhthoff, Graefe-Saemisch.
Marburg, Handbuch d. Neurologie.
Mathilde Windmüller, Über die Augenstörungen bei beginnender Multiple Sclerose, Inaug. Diss., Marburg, 1910.
de Lapersonne et Cantonnet, Neurologie Oculaire.

the cases) and is often the first symptom which precedes the other symptoms by years. Visual disturbances in multiple sclerosis are more frequent than in any other nerve disease, tumor of the brain excepted. When the inflammatory process in multiple sclerosis invades the optic nerve, there are symptoms of a retrobulbar neuritis. The proximity of the inflammatory focus to the papilla determines the ophthalmoscopic picture which is either an optic neuritis or an atrophy particularly of the temporal segment.

The **opthalmoscopic changes** are: (1) incomplete optic atrophy, usually out of proportion to the visual disturbance wherein lies the greatest difference from toxic amblyopia; (2) optic neuritis; and (3) choked disc. In one-third of the cases the optic nerve lesion is one-sided. The visual disturbances may be caused by a lesion situated in the optic tracts without ophthalmoscopic changes.

The onset is often sudden, like an acute retrobulbar neuritis in nasal sinus disease with rapid loss of sight. The condition may exist for some time before extending to the macular fibres, when the patient will first notice a serious defect in sight. In 50 per cent. the progress, however, is gradual. Characteristic is the fleeting disturbance of sight, it can entirely recover, though ophthalmoscopic changes remain. In the very severe cases there is improvement, in the slower cases the amount of sight varies, relapses occur, but the cases never go on to blindness. This acute retrobulbar neuritis was formerly often regarded as "rheumatic." An acute optic nerve affection with rapid development and as rapid improvement occurring in the young who were previously healthy, without known cause, and especially with no signs of lues or an intoxication, should suggest beginning multiple sclerosis.

The **field** also shows a variety of changes, of which a central scotoma is the most frequent. To this may be added some peripheric contraction of the field, or the periphery may alone be involved. The scotoma is usually bilateral and **relative** rather than complete, and therefore easily overlooked. It occurs in about 50 per cent. of the cases. Peripheric contraction of the field with a normal center was observed in 6 per cent. Combination with hysteria must be remembered; this can cause peripheric contraction of the field.

A frequent symptom is nystagmus which is either continuous or appears in the extreme positions. True nystagmus occurs in 12 per cent. The terminal nystagmus occurs in other nerve diseases and sometimes normally in healthy persons.

The **ocular muscles** are affected in 46 per cent. of the cases. The paralyses are slight, incomplete, and may precede the other symptoms. The VI nerve paralysis is most frequent, and isolated branches of the III nerve (ptosis) are not infrequently affected, paralyses of the associated (convergence or divergence) ocular movements have been reported. One-sided incomplete ocular muscle paralysis of usually transient character is typical. There are no pupillary disturbances.

Bodily exertion and fatigue cause distinct diminution of vision when an optic lesion is present. Uhthoff was the first to describe this phenomenon and it is often referred to as Uhthoff's symptom. A peripheric green staining of the cornea has also been described (Kaiser, Fleischer, and Salus).

Syringomyelia.—This disease consists in the formation of cavities which occur in the gray substance and in the central portion of the spinal cord and in the medulla. It usually runs a chronic and progressive course. Ocular symptoms occur when the upper part of the dorsal cord, the cervical cord or the medulla are involved. The optic nerve changes consist in optic atrophy, optic neuritis, or papillœdema. In most cases where there had been a progressive simple atrophy with blindness a complication with tabes or paresis was present. The association of syringomyelia with tabes is not an infrequent one, and tabetic eye disturbances are not uncommon. Optic neuritis or papillœdema must also be regarded as due to a complication either in the form of hydrocephalus or tumor or meningeal conditions. Some authors have laid particular weight upon the presence of a concentric contraction of the visual field without pathological changes in the eye grounds. This symptom, according to Uhthoff, must be regarded as due to complicating hysteria. Paralyses of the ocular muscles are not infrequent. The one most frequently found is an abducent paralysis usually one-sided, rarely bilateral. This is explained by the fact that the abducent nucleus is the one which is situated the furthest down. The spinal process sometimes extends to this location, while the region of the trochlear and oculomotor nuclei is not involved In some of the cases the anomalies of the ocular muscles are due to a complicating tabes, paresis, etc. Nystagmus and nystagmic movements in the terminal positions are relatively frequent in syringomyelia. The pupils frequently show a difference in size. This has been found present in 25 per cent. of the cases and is generally due to a disturbance of the sympathetic nerve.

The symptom of contracted pupil is of great diagnostic importance for syringomyelia as this sympathetic disturbance occurs much more frequently in this condition than in any other disease of the nervous system. The other pupillary symptoms are usually due to a complication with tabes, etc. The trigeminal nerve is frequently involved and is often the only cranial nerve involved. The form in which the sensation is disturbed is a peculiar one, inasmuch as it shows the segmental character indicating a central process. The eye, the conjunctiva and the eye lids are much less frequently involved than the other parts of the face.

Diseases of the Vertebræ.—When the vertebræ of the lower cervical and the upper dorsal cord are affected with involvement of either the cilio-spinal center or of the eighth cervical and the first dorsal roots, along which the oculo-pupillary fibres of the sympathic pass from the spinal cord to the sympathetic ganglia, the ocular fibres of the sympathetic nerve are paralyzed. The most important symptom is a contraction of the pupil with preserved reaction to light and a narrowing of the palpebral fissure. The eye ball does not sink back unless the sympathetic paralysis is an old one. This sympathetic paralysis is usually one-sided. Optic-nerve changes have been described in diseases of the vertebræ. They are very unusual and according to Uhthoff are due to cerebral complications.

HYSTERIA[1]

The symptom-complex of hysteria is an unusually complicated one. No anatomical cause for hysterical symptoms has ever been shown at autopsy, and there is no anatomic change in the eye ground in hysteria which can be brought in any connection with the disease or with hysterical amblyopia.

Hysterical symptoms are not in frequently associated with organic nerve disease; particularly must the connection with multiple sclerosis be remembered, where the eye symptoms, such as changes of the optic nerve with ophthalmoscopic findings, are often the first, preceding the others for many years. Another organic disease which may be associated with hysterical eye symptoms is sclerosis of the cerebral arteries. Traumatic lesions of the eyes with pathological changes in the eye grounds (fracture of the base of the skull with involvement of the optic or other ocular nerves, central retinitis from dazzling, injuries to the occipital lobe, etc.) may later become complicated with hysterical functional disturbances in the form of traumatic neurosis.

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[1] Uhthoff, Graefe-Saemisch, Vol. IX, 3, p. 1592.
de Lapersonne et Cantonnet, Manuel de Neurologie oculaire, p. 318.
de Schweinitz, in Posey and Spiller, The Eye and Nervous System, Lippincott, Phila., 1906.

Hysterical Blindness.—Bilateral amaurosis is less frequent than monolateral in hysteria. In most cases the bilateral hysterical amaurosis occurs suddenly just as it is apt to disappear suddenly. The duration of the blindness can be divided into: (1) the transitory form lasting for a few days; (2) conditions lasting for two to six weeks; (3) long continuous ones lasting from months to years. The attacks of blindness may relapse and be intermittent. In general a single attack is the more frequent and it lasts for a short period, from two to six weeks. Seventy per cent. occur in women, generally under the thirtieth year. As regards the exciting cause, there apparently is no association with hysterical attacks, though it frequently follows psychical disturbances which have existed for some time. A trauma, bodily or psychic in nature, may be the inciting factor. Injuries of the eye and of its surrounding parts, even if they are slight in nature, may cause hysterical amaurosis. Operations not only on the eye but on other parts may also be a cause. Rarely the amaurosis begins spontaneously; as these cases are not frequent, particular care must be exercised against overlooking organic complications on the part of the nervous system, such as multiple sclerosis or cerebral lues.

Wissmann,[1] in a study of Uhthoff's cases and of the literature found in 140 cases of hysterical amaurosis, hysterical changes of general character present in 78 and ocular symptoms in 50. The general symptoms varied from circumscribed island-like areas of anæsthesia to complete sensory hemianæsthesia, from partial paresis to complete hemiplegia, from globus hystericus to the marked picture of pseudo-meningitis. In monolateral amaurosis the general symptoms were usually on the same side. Among the ocular disturbances, changes in the external ocular muscles were present in 27 out of 50 cases, the internal muscles were affected in 34, in some both were affected. In general the other changes consisted in blepharospasm, pseudoparalytic ptosis, spasm of convergence and deviation of the eyes in various directions.

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[1] Augensymptome bei Hysterie, Vossius'sche Beitr. zur Augenhlk., Marhold, Halle, 1914.

The pupillary reaction in hysterical amaurosis is preserved, though the size of the pupil varies; anisocoria may be present and the pupillary reaction be retarded.

The diagnosis of hysterical amaurosis is sometimes extremely difficult in the absence of other symptoms, especially when it occurs as the only symptom in children and occasionally in adults. The cases of hysterical amaurosis which do not recover are very infrequent. Hysterical amaurosis is to be regarded as a psychical one. The retina as well as the visual center perceive the visual impression but this picture is neutralized unconsciously by the mind. It is often difficult to decide between hysterical amaurosis and simulated blindness.

The **change in the visual field** which has the greatest diagnostic significance in hysteria is the uniform concentric contraction. Disturbances of sensation are a frequently associated symptom of this defect of the field. If they occur in the characteristic form of hemianæsthesia including half of the head and the sensory organs of that same side (hearing, smell and taste), they present a typical symptom-complex of hysteria. The sensory disturbances need not be truly hemianæsthetic, but may be partial, zone-like, or island-formed anæsthetic areas of the skin. In general, concentric contraction of the visual field is usually complicated with other sensory disturbances; when these are absent, the question of simulation or of an organic disease must be considered. The concentric contraction is usually bilateral and is apt to be more marked on the side where the sensory disturbances are the greater. The relation of concentric field contraction to sensory disturbances of the eye has been frequently discussed. One author believes that concentric contraction is always proportional to a disturbance of sensation of the eye, particularly of the cornea. This is denied by Binswanger. Nevertheless this is a frequent complication, and according to Uhthoff a sensory disturbance of the conjunctiva is more frequent and more easily demonstrable than that of the cornea. Most authors regard distinct concentric contraction of the field as of decided diagnostic value in hysteria. The concentric contraction varies in degree; this variation determines the functional character. A characteristic is that orientation is not lost. The linear extent of the field at various distances remains the same, "tubular character," instead of being proportional to the distance.

A central scotoma has been described by some in hysteria, but it does not belong to the clinical picture of hysterical amblyopia, and suggests a complication with multiple sclerosis. Typical homonymous hemianopsia is not a symptom of hysteria.

In hysteria the concentric peripheric contraction of the field is accompanied with a contraction for **color perception** which is described either as achromatopsia or a dyschromatopsia. A complete loss of color perception is very unusual. Dyschromatopsia means a simultaneous contraction of color perception. The color outlines are crowded together but preserve their physiological sequence or their order is changed; sometimes the color outlines interlace. A number of other types of field defects have been described[1] which are not typical for hysteria but are found rather in neurasthenia and traumatic neurosis. One of these is the "fatigue" field in which the abnormal fatigue of the patient can be demonstrated by determining the meridians in the order of a clock dial and the outline of the field will correspond to a spiral curve with one end nearest the point of fixation. The other form is Foerster's shifting field ("Verschiebungstypus"), which depends on the fact that the appearance of an object makes a stronger impression than its disappearance.

An occasional symptom of hysteria is that the outer world appears in a particular color.

[1] de Schweinitz, p. 614.

Ocular Muscles.—The disturbances in the motility of the eye in hysteria are often complicated because they are spastic in nature. Individual muscles are not usually affected, but associated or disassociated ocular movements suffer, in other words, movements which are under the voluntary influence of the patient. The diagnosis of an hysterical ocular-muscle anomaly depends greatly on the presence of other hysterical manifestations, as it is rarely the only symptom of hysteria. According to Mauthner, the frequency of hysterical ocular paralysis is in direct proportion to the possibility of a voluntarily produced lesion. A true paralysis is extremely uncommon. Uhthoff has not seen a single definite case, though there are a number of observations in literature which apparently prove the possibility of an isolated ocular paralysis. In any case, they are very rare and this holds especially true for ptosis which in only individual cases can be regarded as a true paralysis, as it is usually due to a spastic condition on the part of the orbicularis. Conjugate deviation of the eyes has been observed during an attack. Paretic disturbances of the disassociated ocular movements, particularly of convergence, is sometimes an hysterical symptom. Typical oscillating nystagmus can not be regarded as a symptom of hysteria.

It is frequently difficult to distinguish between a spasm and a real paralysis of an ocular muscle. Spasmodic conditions of the orbicularis are common and vary in degree from slight fibrillation to marked blepharospasm. The orbicularis, according to Wissmann, was affected in 66 per cent. of the eye changes. This spasm is either one- or two-sided, and may be intermittent or permanent. It is important, in this connection, to observe the state of the other muscles of the face, for spasm of the facial muscles is frequently combined with tonic contraction of the orbicularis. In hysterical children blepharospasm is frequent and may be the only symptom. The development of the blepharospasm is generally a gradual one following inflammatory external diseases of the eye. The ptosis is spastic (pseudo-paralytic), and rarely paralytic. Charcot and Parinaud were the first to clearly describe pseudo-paralytic ptosis. The condition is characterized by a lowering of the eye brows with obliteration of the frontal ridges, the palpebral fissure is not entirely closed, and the margin of the lid has a wavy form. The upper lid hangs down in concentric folds of skin, and a slight elevation of the lower lid is sometimes observed.

Spastic conditions of the external ocular muscles produce complicated conditions and the double images are difficult of interpretation. Among the spasms in hysteria those of the internal recti are the most marked. In this convergent strabismus there is no defect in motility though diplopia can be demonstrated. The spastic hysterical convergent strabismus is either continuous or intermittent. Monocular diplopia and polyopia in hysteria are probably due to incomplete spasm of the accommodation. Spasm of the accommodation is a frequent accompaniment of spasm of the outer muscles (convergence) and amblyopia. Macropsia or micropsia is often an hysterical symptom which, according to Parinaud, is sometimes associated with hysterical amblyopia and is more frequent than monocular diplopia.

Some authors state that the pupils in hysteria are wider than normal. It was formerly believed that the reaction of the pupils to light in an hysterical attack was preserved; but a number of authorities have shown that the light reaction may be affected or abolished under certain conditions in hysteria.

Among the sensory disturbances in hysteria the trigeminal nerve is frequently involved and circumscribed areas of anæsthesia which include the eye have been found present, though there is a normal diminution in the conjunctival and corneal reflexes in advanced years. The combination of hysterical amblyopia, concentric visual field contraction with disturbances of the corneal and conjunctival reflexes is frequent, though not the rule.

EPILEPSY

Genuine epilepsy must be distinguished from the symptomatic which can be caused by a variety of intracranial lesions. Examination of epileptics during attacks have given either negative findings, or venous stasis of the eye grounds together with general cyanosis of the face. Uhthoff[1] has never found distinct contraction of the retinal vessels with pallor of the optic nerves. In his experience pathological eye changes in genuine epilepsy are infrequent. Inflammatory changes at the disc are not a symptom of genuine epilepsy. Subjective disturbances in the aura before the epileptic attack have been frequently described. The functional concentric contraction of the field, particularly in connection with an epileptic attack, is not an unusual symptom. The refractive conditions of epileptics are not particularly different from those of normal individuals. Uhthoff noted only the marked degrees and received the impression that perhaps the high grade congenital refractive anomalies are somewhat more frequent in epileptics than in the normal.

The opinions of certain authors on the favorable effect of the correction of muscular imbalance in epileptics are exaggerated and too optimistic. It is quite easily understood that very nervous epileptics suffer more from uncorrected refractive anomalies and disturbances of motility than others do; on the other hand, the therapeutic importance of their correction in preventing the onset of epileptic attacks has surely been over-estimated.

Spasmodic contraction of the eye muscles at the beginning of the epileptic attack is a frequent symptom, particularly the lateral deviation of the eyes often accompanied with nystagmic oscillations. During the epileptic attack the pupils are reflexly immobile and dilated. It has been generally accepted that a distinct symptom of epilepsy was the abolition of the pupillary reaction during an attack, whereby it could be distinguished from an hysterical attack where the pupillary reaction is preserved. A number of authors have shown, on the other hand, that exceptionally the pupillary reaction in an hysterical attack may be lost and at the same time it may be preserved in the epileptic attack.

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[1] L. c., p. 1576.

At the beginning of the epileptic attack the pupils become widely dilated, then contract, and during the attack do not respond to light nor upon any attempted convergence of the eyes. Sensory stimuli are not capable of dilating the pupils during an attack. During the intervals the pupils of the epileptic do not show any particular change. Uhthoff thought that in some of his patients the pupillary reaction to light was perhaps an exceptionally active one and that the contraction was promptly followed by a certain amount of dilatation.

TRIGEMINAL NEURALGIA (Tic Douloureux)

The V nerve is the one most frequently affected by neuralgia, particularly in persons with neuropathic tendencies. The neuralgias are generally due to some organic cause, most frequently a neuritis of the V nerve or of the Gasserian ganglion, caused either by a poisoning from alcohol, tobacco, lead, diabetes, gout or an infection such as influenza, syphilis, malaria, or rheumatism, or from disease of the teeth and of the nasal sinuses. There is a form which is regarded as senile and arteriosclerotic.

The attacks are often preceded by curious sensations of itching, creeping, though most frequently they develop suddenly. The sensibility is never normal; there may be anæsthesia, particularly in the old and in the severe cases, but more frequently the skin, the conjunctiva and the cornea are distinctly hyperæsthetic. Photophobia and slight blepharospasm are practically always present in ophthalmic neuralgia. There are also vasomotor disturbances. In addition to the point of painful pressure at the supraorbital foramen, there may be a tender point somewhat nearer the superior oblique pulley and another at the lobe of the nose, and one at the frontal and parietal eminences. Between the attacks the patient usually has no pain, possibly some sensations of heaviness and of heat, so that he will avoid every sudden movement, very bright lights, ocular fatigue and cold.

In tic douloureux, in addition to neuralgia, motor troubles are marked. Instead of there being a slight blepharospasm, the blepharospasm is intense, the pain is atrocious and the prognosis is worse.

The affection is usually one-sided and one or two branches of the V nerve are involved. The pain is often terrific. It can continue for several minutes and repeat itself for hours. The pain is frequently associated with tearing of the affected eye and increased nasal secretion. There may be spasms in the muscles of the face. In two-thirds of the cases the first branch (ophthalmic neuralgia) is involved. The pain is situated over the eye, radiates backwards, there is a pressure point at the supraorbital foramen. There are forms of neuralgia which are localized to the eyeball (ciliary neuralgia). The severe pain is then either in the eyeball or directly back of it, with tearing, redness of the conjunctiva and photophobia. The writer has observed so severe a case of ciliary neuralgia that the eyeball had to be removed.

The cases of neuralgia vary in severity. There are those which recover after a few weeks, and some which continue for years. The severe neuralgias are particularly frequent in advanced years.

HERPES ZOSTER OPTHALMICUS (Ophthalmic Zona)

Herpes zoster, notwithstanding the associated corneal anæsthesia, is an irritative symptom of the V nerve. The attack is usually preceded by neuralgic pain accompanied by general malaise, nausea, fever and loss of appetitie. An eruption then develops suddenly in the form of erythematous plaques on an œdematous surface which is most marked in the upper lid and the eye is sensitive to light. Yellowish vesicles appear on these plaques after a few hours and often unite to form large blebs. They are situated along the passage of the nerves, anteriorly forming vertical lines on the forehead, somewhat diverging as they pass backward. A characteristic is that this eruption never crosses the median line and in the great majority of the cases it is one-sided. Occasionally the eruption turns purulent or hemorrhagic. After a few days the vesicles dry up, dark crusts form which leave white scars. The pain persists for a long time and is accompanied by a curious anæsthesia. Herpes zoster is now regarded as an acute infection of the spinal ganglia by an unknown infectious agent. A toxic cause is also accepted as it has been observed in poisoning with coal gas, gout and diabetes.

In 60 per cent. of the cases there are ocular complications, the conjunctiva being affected most frequently, in the form of vesicles with discharge and anæsthesia.

Complications on the part of the iris are less frequent. The iritis may be either mild or serious when secondary to corneal lesions. Reduced intraocular tension is not infrequent. The affections of the optic nerve and of the ocular muscles should be regarded as due to the same cause as produces the zona.

According to Fage,[1] the paralytic symptoms may affect the entire III nerve or only produce ptosis or mydriasis. Involvement of the IV and VI nerve are exceptional, though all ocular muscles may be involved. In these cases a slow recovery is the rule.

The corneal complications are the most important. They occur in 35 per cent. of the cases. One of these forms is interstitial keratitis, usually localized to the central parts of the cornea. The most frequent corneal complication, however, are ulcers following vesicles. They are accompanied by corneal anæsthesia. After a number of weeks the corneal lesions usually improve, leaving opacities. Sometimes the ulcers become infected and any of the complications of a severe corneal ulcer result. It is stated that ocular complications follow an involvement of the nasociliary nerves so that they are always accompanied by an eruption on the side of the nose. This is described as "Hutchinson's law," though its correctness is denied by some. After an attack of zona persistent supraorbital neuralgia may ensue.

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[1] Fage, Rec. d'ophthal., p. 209, 1909.

OPHTHALMIC MIGRAINE[1] (Scintillating Scotoma)

Ophthalmic migraine is that form of migraine which is preceded by pronounced visual disturbances. The visual disturbances vary in degree and in kind, though no specific ocular changes have ever been observed. The malady usually occurs in middle life. It may run a very protracted course. If the first attacks begin in advanced age, organic disturbances (diseases of the cerebral vessels, nephritis) should be considered. The attack usually consists, after certain prodromal symptoms or without these, of a sudden disturbance of sight, usually of a scintillating character, lasting from a number of minutes to a half hour, then gradually disappearing, followed by headache, which may be accompanied by nausea and vomiting.

The visual disturbances can be divided into two groups: (1) the simple transitory disturbance; (2) disturbances of a scintillating and spectral character. There are varying grades found in the first group, from slight fogging to complete amaurosis. This form may attack one or both eyes. In some cases only a part of the field is involved (hemianopsia); very rarely there is transverse hemianopsia, and Antonelli has described a central scotoma which enlarged to occupy the entire visual field. The second group is very much more frequent; in this the striking symptom is the scintillating, sparkling, often spectral character of the visual disturbance. The fog may have a continuous motion, like heated air over a stove, in other cases there are symptoms of light, or some form of figures with spectral colors which surround the margin of the scotoma or penetrate the scotoma. Scintillating scotoma generally begins with a brightly burning point appearing in a circumscribed part of the field, usually near the middle line. It enlarges, takes on a zig-zag, fortification-like figure in constant motion and in colors, extends gradually to the periphery and diappears in from a few minutes to a half hour. Sometimes the onset and the progress may be reversed from the periphery to the center. Both eyes are usually attacked though the phenomenon may occur in one eye. The entire field is more frequently involved than half of the field. The scintillating symptoms generally precede the scotoma, though the reverse condition has been described. The form of visual disturbance sometimes varies and the same patient may have a number of attacks of simple obscuration and then an occasional complicating scintillating scotoma.

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[1] Flatau, Lewandowsky Handbuch d. Neurologie, Vol. IV, p. 361.

After the scotoma has disappeared, the headache begins. This headache is usually very intense, rather more severe than in simple migrain. It is frequently accompanied with nausea, vomiting and vertigo and the attacks may last from one to three days. Its site corresponds to the affected eye, though in some cases it is situated at an entirely different part of the head; if the visual disturbance is hemiopic, the headache affects the opposite half of the head.

Exceptionally the headache begins the attack and the visual disturbance follows, or the visual disturbance may be present without migraine (formes frustes). There is a form of migraine with associated symptoms of sensory epilepsy (migraine accompagnée of Charcot) in which paræsthesias occur in half of the body. A further complication is aphasia of motor character which may be either complete or relative. In the latter case the patient is able with the greatest difficulty to articulate a few words. This condition continues from a few minutes to several hours. The course, in general, is that the attacks disappear without leaving marked defects, though in some cases there are distinct signs of mental involvement, loss of memory, mental slowness, diffuseness, the affected individual may become eccentric and irritable. Sometimes the symptoms become permanent, as for instance the aphasia or the visual disturbances.

The examination of the retina, particularly during the attacks, has always given negative findings. The correction of refractive errors or of muscular imbalance has not exerted any influence on the attacks.

OPHTHALMOPLEGIC MIGRAINE

In this form of migraine the attacks are accompained by paralysis of the ocular muscles. Up to the present time 97 cases have been reported. This form was described by Möbius as "periodically recurring oculomotor paralysis" It occurs in cases where from the early youth or childhood there are recurring attacks of oculomotor paralysis accompanied with headache and vomiting. Möbius separated these cases from true migraine, while Charcot believed the ocular paralysis to be but a symptom of the migraine, and gave the syndrome its name of ophthalmoplegic migraine.

This condition occurs usually in middle age. The attack consists in migraine accompanied with ocular paralysis. There is usually a one-sided severe headache localized to the neighborhood of the eye, the forehead, and the temple. After the pain has existed from one to two days or more, the paralysis usually ensues in the distribution of the oculomotor nerve and the headache then ceases. Sometimes the patients will have an attack of pain lasting for weeks or months before the ocular paralysis appears and relieves the pain.

The prodromal headache may vary in the same individual and sometimes occurs at the same time with the ocular paralysis. It is nearly always one-sided and on the same side where the ophthalmoplegia develops. The headache is usually severe and accompanied with nausea and vomiting. In most cases this migrainous symptom-complex precedes the ophthalmoplegia, in other cases the symptoms consist in a sense of depression, restlessness and scintillating scotoma. The last symptom has been known to occur for days before the ocular paralysis. There may be epileptic-like symptoms, psychical disturbances or disturbances of the sympathetic nerve.

The ocular paralysis, in most cases, affects the III nerve, rarely the VI or the IV. Sometimes there is a combined lesion of two or three ocular nerves. The oculomotor paralysis is usually on the side of the headache. It is usually a complete one, both external and internal branches being involved. The eye is turned out or out and down, with restricted motility, ptosis, dilated pupil and paralysis of accommodation. This complete III nerve paralysis appears in the first attack and is repeated in all subsequent attacks. Sometimes in the first attack a few of the muscles only are affected, and later the paralysis becomes complete. In a small proportion of cases the III nerve paralysis is not a complete one or the paralysis may extend without becoming complete or the incomplete involvement may vary in the same person. Up to the present time two cases have been reported in which exclusively the IV nerve was affected, and four where the VI nerve was involved. Sometimes two or three of the ocular nerves may be involved in the same patient. There are three cases on record in which all three of the ocular muscles have been involved.

The duration of the ocular paralysis varies in the individual attacks—generally it lasts only one or two days, though it has lasted for a number of weeks or months. As a rule the ocular paralysis disappears completely or slight residual symptoms remain. The muscle gradually recovers in from several weeks to three months. In the early attacks complete recovery may occur, but later some of the muscles are apt to become permanently paralyzed. The period between the attacks varies; frequently the intervals are long, attacks occurring only once or twice a year, sometimes at intervals of several years. The longest intervals have been between six to ten years.

Complications have been described on the part of the other cranial nerves (the V nerve) and of the optic nerve (hyperemia, optic atrophy, scintillating scotoma, visual field disturbances). The most frequently described complication is the scintillating scotoma and the hemianopsia, which may be accompanied with other disturbances seen in associated ophthalmic migraine. It is remarkable that these patients who suffer from ophthalmoplegic migraine have sometimes in earlier life suffered from simple migraine. Both forms of migraine may occur early in life, as has been stated, and the forms may vary in the same patient, though it is very rare that attacks of ophthalmoplegic hemicrania are later followed by simple migraine. Ophthalmoplegic migraine is rarely observed without intermediate attacks of simple migraine. This shows that there is a close relation between these two forms, as has been particularly dwelt upon by Charcot.

Spicer and Ormerod[1] found that in recurring one-sided headache, with vomiting and prostration the slight attacks pass off in a few hours, the more serious ones last several days accompanied with sudden and more or less complete III nerve paralysis. In the early attacks recovery takes place, then with recurring attacks the function of the III nerve becomes permanently impaired. These authors have collected 36 cases of recurrent paralysis of ocular nerves; usually of the III sometimes of the VI, mixed or bilateral. No other migraine symptoms were present except headache. Karplus[1] found at post-mortem a fibroma completely involving the nerve.

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[1] Recurrent Paralysis of the III Nerve, T. O. S., 96.

TIC[2]

Tic consists in momentary lightening-like contractions limited to a small group of muscles, chiefly of the face. These contractions apparently occur without cause. A tic limited to the eye lids is very frequent. It occurs generally in the form of clonic, rarely of tonic spasms; in the latter case, after an abnormally long contraction of the eye lids the lids are kept half closed. In all of these ocular tics, conditions of irritation of the eye have usually been the original cause. Blepharospasm occurs as clonic convulsions in the form of constant blinking or the contraction is more permanent when the eyes remain closed for some time. Orbicularis spasm occurs in chorea, migraine, epilepsy and hysteria.

Convulsive tic in hysteria generally affects both eyes; both eyes suddenly close and then open just as suddenly. The spasm can be relieved by pressure on some points in the V nerve. Traumatic neuroses after slight injuries to the head are sometimes followed by functional orbicularis spasm. Blepharospasm may be a senile symptom.

Spasm of the facial nerve is caused reflexly by painful affections of the conjunctiva and of the cornea, by carious teeth or any other affection of the V nerve, and the condition may be associated with tic douloureux. Given a disposition to a condition of this kind, any local irritant will produce the tic. Neuropathic individuals are particularly liable. There is a form of contracture in the distribution of the facial nerve after facial paralysis which should be separated from true facial spasm. Facial spasm is usually one-sided and clonic, though it may be combined with tonic contractions or these two forms may alternate. When the orbicularis is tonically contracted the skin of the forehead and of the adjoining parts of the nose and mouth is wrinkled, causing distinct furrows in that half of the face.

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[1] Zur Kenntniss d. periodischen oculo-motorius Lähmung, W. kl. W., 1895, Nos. 50–52.

[2] Fritz Mohr, Lewandowsky Handbuch. d. Neurologie.
Oppenheim's Lehrbuch, p. 1641, 1914.

Clonic contractions vary in intensity from being hardly distinguishable to active grimaces. The condition usually comes on in paroxysms, influenced very much by mental and physical unrest. In many cases, particularly in blepharospasm, pressure points exist in the distribution of the V nerve and where compression arrests the spasm.

In addition to the facial spasm, symptoms of other nervous diseases are frequently present, such as hysteria, epilepsy, hemicrania, psychosis, etc. The course is usually chronic, though intermittent, and the prognosis is not favorable. In most cases there are relapses and the condition is more or less permanent.

SYMPATHETIC DISTURBANCES

Differences in the size of the pupil are often due to a lesion of the sympathetic nerve and it is important in this connection to be on the lookout for other ocular sympathetic symptoms. These consist in ptosis of moderate degree accompanied by miosis. The upper lid not only droops but the lower lid is somewhat raised, due to a paresis of the smooth muscles in the eye lids. After the paresis has existed for a certain length of time, the eye ball sinks back and the instillation of cocaine causes but little dilatation of the narrowed pupil. This is particularly noticeable on instilling cocaine in both eyes. The narrowed pupil reacts to light and to convergence. The difference in pupils is more noticeable by reducing the illumination in which the normal pupil dilates, while the narrowed pupil practically does not change.

In addition to the ocular signs there are vasomotor disturbances of the same half of the face. Horner[1] showed that there are two periods in a sympathetic paralysis during which the pupil remains contracted and the lid-opening narrowed. In the first period there is increased redness, increased perspiration and temperature on the affected side. In the second period the paralyzed side is pale, there is an atrophy of tissue (enophthalmus) reduced temperature and and absence of perspiration. These symptoms generally go under the name of Horner's syndrome, to which the French add the name of Claud-Bernard. To repeat, the characteristic paralytic oculo-sympathetic symptoms are: miosis, slight ptosis, enophthalmos, vasomotor disturbances and hypotony of the eye.

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[1] Über eine Form von Ptosis, Kl. Monatsbl., f. Augenhlk., 1869.

One-sided sympathetic paresis usually means a struma in which the size is not as important as the position and the toughness of tumor tissue. The condition is also produced by affections of the apices of the lungs, tumors of the cervical glands, scars in this region, aneurysms of the carotid and carcinoma of the œsophagus. If the sympathetic paresis is associated with paralysis of the arm, the condition is regarded as a root lesion, in contradistinction to Erb's plexus affection. In other cases it indicates a fracture of the cervical vertebræ. Sympathetic paralysis with crossed body symptoms and uncrossed trigeminal disturbances indicate the site of the affection to be below the pons on the side of the sympathetic symptoms. Sympathetic paresis may occur in syringomyelia, hematomyelia, and cervical myelitis.

If the paresis is not distinct, the cocaine test can be applied. In each eye the same amount of cocaine, one drop of a 5 per cent. solution is instilled. After fifteen minutes the normal pupil and palpebral fissure are dilated; the paretic is only slightly changed or not at all.

One-sided sympathetic irritation is very much less frequent than the sympathetic paralysis. It is associated with dilated pupil, dilated palpebral opening, exophthalmos, occasionally with one-sided Graefe symptom. The causes are the same as those that lead eventually to a paralysis.

Heterochromia has been brought into relation with a sympathetic paralysis. Mayou[1] has reported on heterochromia iridis associated with paralysis of the sympathetic nerve in early life. There was drooping of the upper eye lid and miosis. The history of a difficult forceps labor suggested a birth injury. Bistis[2] has made a similar observation and has confirmed the association of sympathetic paralysis and heterochromia by animal experimentation.

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[1] Mayou, T. O. S., 1910, p. 196.

[2] Bistis, Arch. of Ophth., Vol. XLIV, p. 433, 1915.

III. DISEASES OF GLANDS WITH INTERNAL SECRETION

EXOPHTHALMIC GOITRE[1] (Basedow's Disease)

This is a disease which usually occurs in the third or fourth decade and affects women more frequently than men. There is a distinct heredity and symptoms of nervousness or hysteria generally precede, though a psychical or mental shock often serves as the actual cause. There may be a history of bodily over-exertion, pregnancies, or illnesses before the attack. The three cardinal symptoms are: (1) tachycardia; (2) struma; (3) exophthalmos. The first is the most important and the earliest symptom: the pulse rate is 120 to 140, increasing on excitement. The thyroid tumor comes on later; it may not be apparent though gland is diseased. Exceptionally exophthalmic goitre develops in the other thyroid diseases.

Though the eye symptoms in exophthalmic goitre occupy a prominent place, they do not permit any deduction as to course, severity or outcome; the other symptoms on the part of the heart, nervous system and metabolism are more important.

Exophthalmos.—The exophthalmos is generally bilateral, though at first it may only affect one side, and then it is usually the left. It varies in grade and at times. Absence of exophthalmus is not very unusual (formes frustes). The eye can be pressed back into the orbit. Pressure on the facial veins will cause increased exophthalmos. An abnormal distention of the orbital veins is probably present. The eyes are pushed straight forward and their motility is not restricted. This symptom is usually the last cardinal symptom to develop and it is absent in 20 per cent. (Sattler). It also stands in no relationship to the severity of the affection.

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[1] Hans Eppinger, Handbuch d. Neurologie. Lewandowsky, Berlin, 1913.
Oppenheim, Lehrbuch d. Nervenh'lk. 1914
Sattler, Graefe-Saemisch-Hess.

It is necessary to distinguish in the symptoms of exophthalmos between a protrusion of the eye ball and a widened palpebral fissure. This symptom which can be best recognized on profile view does not necessarily mean an enlarged palpebral opening. It is sometimes more pronounced on one side than on the other. The exophthalmometer has shown that a difference is quite frequent, particularly in the beginning of the disease. It is not possible to find a connection between the protrusion which is more marked on one side with a struma which is more developed on the same side, though if half of the struma has been removed by operation it is not unusual to find that the protrusion on the operated-on side has disappeared.

Sattler has collected 109 cases of one-sided exophthalmos. The degree of the protrusion varies and is not parallel to the intensity of the general symptoms. If it varies, there is an association with the intensity of the palpitation and the general condition. The exophthalmos is often a late symptom and remains as the last sign after recovery. In severe cases the protrusion may be so marked that a covering of the sclera is impossible even in sleep. Luxation of the eyeball is a very unusual symptom. The exophthalmos, according to Sattler, is the result of œdema of orbital tissues following vasomotor disturbances. In favor of this view is the frequently observed œdema of the eyelids and the findings at the operation of resection of the outer margin of the orbit after Dollinger.

Widening of the Palpebral Opening (Dalrymple).—This is due to an abnormal retraction of the upper lid and is often confused with protrusion. The wide opening of the lids is more properly spoken of as Dalrymple's sign. The wide palpebral opening is not the direct result of the protrusion of the eyeball. This symptom is also not equally present in both eyes. It may be present in only one side and it also varies greatly in its intensity. The wide opening shows a portion of the sclera both above and below the cornea, and is frequently a very early symptom being present without protrusion. It is caused by contraction of the unstriped tarsal muscles.

Graefe's Symptom.—This is a remarkable disturbance of the association in movement of the upper lid, a lagging behind of the upper lid on looking down. In looking upwards the association is also disturbed as the upper lid moves independently and more actively than the eye ball. In the mildest forms the rim of sclera is first seen on looking downward. This symptom occurs in individuals who do not suffer from Basedow's disease. Like the protrusion, it may be present on one side independent of whether the protrusion is one or both-sided. This symptom is due to increased tonus of the levator palpebræ superioris (Möbius).

Jessup and Edmond believe to have observed an increased gaping of the lids after adrenalin or cocaine. This has not been confirmed by Sattler. Sattler now has accepted the theory of Möbius, who believes that there is an exaggerated tone of the muscles which keep the eye open, which is permanent in Basedow's and transient in conditions of excitement. As the muscles which are enervated by the sympathetic are not concerned, there must be an increased tone of the elevator of the eyelid. Gifford found that the turning of the lid under these conditions was difficult.

Stellwag's symptom is a lessening of the reflex closure of the lids. In the normal three to ten closures of the upper lid occur in the minute, while in Basedow's disease this may not occur but every few minutes.

Möbius' symptom is the insufficiency of convergence. In Basedow's disease the eyes soon give up the attempt to converge. This does not depend upon the protrusion of the eyes or upon the lid conditions, and indicates an insufficiency of convergence.

Other Eye Symptoms—Mydriasis, nystagmus and disturbances of accommodation are unusual symptoms. In exophthalmic goitre Loewi's phenomenon is often present dilatation of the pupil after instillation of adrenalin. Cases of optic atrophy are of interest, because in animals after thyroid feeding optic atrophy has resulted, and in patients who have been treated on account of myxœdema, with long continued doses of thyroid gland, optic atrophy has been observed. Another symptom is unintentional tearing, which is regarded by some as an early symptom. It has seemed to the writer that the tearing in these cases may be due to a displacement of the lacrymal point as the stretching of the lids from the exophthalmus causes an inversion of the lacrymal points. In severe Basedow's disease paralysis of ocular muscles may occur. Bilateral ophthalmoplegia externa, paralysis of associated movement or of individual muscles have been described.

From imperfect protection, the cornea may suffer from infiltrations which in turn may lead to any one of a number of serious conditions; moreover, the drying of the superficial corneal layer invites infection. It can not be shown that a diminution of the sensibility of the cornea is a frequent symptom. As to the corneal suppuration, it must particularly be insisted upon that the imperfect covering or insufficient tears can not be regarded as the cause for all cases. Both eyes are frequently affected, and the most energetic treatment seems sometimes without avail.

A most serious **corneal complication** is a purulent disintegration of both corneæ. This is seen in cases of unusual severe Basedow, though it has been observed in cases where there was no exophthalmos. Sattler regards this complication as directly due to a toxic action on the cornea. Jessop[1] states that in one case after tarsorrhaphy the corneæ sloughed. There was extreme exophthalmos so that the eyes became easily dislocated. As soon as the corneæ was affected, no treatment had any effect. In the other two cases, corneal necrosis occurred without operation. Twenty-five cases of corneal involvement are tabulated. Nettleship advised thorough suture of the lids, as local treatment was of no avail. Charles H. Mayo recommends in severe corneal complications, sympathectomy and advises against suture of the lids. F. A. Juler[1] favors orbital incision and free tarsorrhaphy to relieve the pressure of the globe against the lids. The writer succeeded in saving a desperate case by suturing the lids together after widely splitting them as suggested by Priestley Smith[2] and making releasing incisions[3] in the skin at the orbital margin.

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[1] Three Cases of Exophthalmic Goitre with Severe Ocular Lesions, T. O. S., 1896.

MYXŒDEMA

One of the characteristic signs of a cessation of thyroid function is changes in the skin. There is a gradual thickening of the skin which is mostly noted in the face and hands, and the eye lids are frequently first affected. The skin becomes dry and inelastic. The thickening, particularly noted below the chin, and the changes in the hands are quite characteristic. A nearly constant symptom is a loss of hair which is noted not only on the scalp but in the eye brows. Groenouw speaks of a quivering of the eye lids on the patient's attempt to close them, which is however not characteristic but is frequently observed in a number of nervous diseases.

There are changes secondly in the nervous system, though these on the part of the cranial nerves are relatively unusual. According to Eppinger, the patients frequently complain of defective eye sight, though there is no characteristic eye lesion. Disturbances in the ocular muscles do not occur, though the patients frequently close their eyes, apparently unintentionally. A contraction of the visual field above has been described, though this may be the result of a prominence of the upper eyelids. Further characteristics consist in slowness of speech, loss of memory and general lassitude.

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[1] T. O. S., 1913, p. 58.

[2] Priestley Smith, T. O. S., 1913, p. 71.

[3] Bishop Harman, T. O. S., 1913, p. 74.

The intelligence gradually deteriorates and hallucinations of the various senses have been observed. The cause is an atrophy of the thyroid gland. A reduction in thyroid activity retards the chromaffine system, while the activity of the pancreas and of the epithelial bodies is increased.

In addition to these disturbances of the lids and of the eye brows, a number of ocular diseases have been brought in connection with myxœdema, such as interstitial keratitis, optic atrophy, optic neuritis and cataract. The proof of this relationship is difficult to bring. Collins[1] reported the case of a woman aged fifty-eight, with skin condition characteristic of myxœdema, who developed an opacity in the cornea, a central grayish haze in the superficial layers which he regarded as a deposit of mucin and which cleared up under thyroid treatment. A very interesting association is that of myxœdema with bitemporal hemianopsia suggestive of the associated enlargement of the pituitary gland with an atrophy of the thyroid. Derby[2] has reported on bitemporal hemianopsia in myxœdema, and finds that only in the rarest cases of myxœdema, cretinism and similar diseases, is there an enlargement of the hypophysis sufficient to cause optic nerve symptoms. Bordley[3] has obtained good results in the treatment of malignant uveitis with thyroid extract without stating that myxœdema was the cause for the eye disease. Dunn[4] believes that hypothyroidism should be considered as a cause in iridocyclitis. The ciliary body, according to this author is particularly sensitive to toxemia and readily responds to thyroid treatment on account of its vascularity. The author gives the extract: 3 grains twice daily for adults. In the treatment of myxœdema thyroid extracts have given good results. Coppez[1], however, warns against the excessive use of this remedy as it has produced optic atrophy.

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[1] Collins, T. O. S., 1907.

[2] Derby, Diseases of the Optic Nerve in Myxœdema, J. A. M. A., 1912.

[3] Bordley, Arch. of Ophth., Vol. XLIV, 1915.

[4] Dunn, Lancet, May 29, 1915.

TETANY[2]

This disease is characterized by trophic disturbances in the hair, teeth, nails, lens and general disturbances of metabolism. The nails are cast off or are extremely fragile. The hair can be completely lost. A sign of tetany in the early period of life shows itself in the teeth. There are defects in the formation of the enamel and in the ossification of the dentine; transverse ridges with round holes are formed. The teeth most frequently involved are the incisors and the teeth directly adjoining. The defect in the teeth, according to Phleps, occurs more frequently than cataract.

A very important trophic disturbance is the development of cataract. This has been demonstrated more frequently in the tetany of adults than in children, and appears in the severe form after struma operation with injury to the epithelial bodies as well as in the tetany of maternity, though it is not unusual to find it in the other forms. In the acute, severe forms it occurs together with changes in the hair and in the finger nails. In the chronic relapsing types it often occurs without these.

Hesse and Phleps' investigations have shown that zonular cataract occurs to an unusual degree in the latent cases of tetany, where acute exacerbations were present only in early childhood. Lenticular changes, according to these authors, occur in juvenile tetany much more frequently than was previously believed. At the same time it must be insisted upon that in these cases the changes need not

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[1] Coppez, Archives d'ophth., p. 656, 1900.

[2] Eduard Phleps, Lewandowsky Handbuch. d. Neurologie.

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be gross and that they do not produce in most of the patients any subjective symptoms. In many cases there are only isolated marginal delicate linear or punctate opacities with however a typical perinuclear distribution. The proper examination requires a dilatation of the pupil. The cataract in some cases is the only prominent change remaining after a tetany which has been recovered from many years previously, and demands notwithstanding a negative anamnesis, a careful investigation for latent signs of insufficiency of the epithelial bodies. The opacities are usually in the form of a zonular or of a diffuse lenticular opacity, more rarely of an opacity of the nucleus or of the anterior polar variety. In general, both lenses are affected. Similar changes in the lenses were observed in experimental animal tetany and occasionally in the new-born of human beings and animals suffering from tetany.

As to the histological characteristic of a tetany cataract, in analogy with the trophic disturbances of the teeth, the opacities of the lens in their formation point to a periodic development, inasmuch as normal lens substance is present between individual layers of opacity. Pineles has drawn attention particularly to the connection of lenticular opacities with trophic disturbances, and this author mentions the frequency with which cataract develops in other diseases of the glands with internal secretion, as in diabetes (pancreas, adrenals). All of these trophic disturbances involve tissues of ectodermal origin.

The rachitic etiology of zonular cataract nevertheless still occupies its dominating position. In the absence of exact neurological examinations, Frank, who has written on the nature of convulsions in zonular cataract, can not exclude latent tetany. Moreover, rickets and tetany are often associated. Hesse and Phleps have shown the exclusive etiological significance of tetany for zonular cataract. They were the first to institute systematic examinations in a large number of patients with zonular cataract. They found in their 43 cases distinct symptoms of tetany in 81 per cent., and if we regard the cases where tetany can not be definitely excluded, this number is increased to 90. They conclude that in zonular cataract and in many varieties of cataract occurring in the presenile age tetany is the cause. They, moreover, believe that the lens may be affected in other form than in the typical zonular form in tetany.

Fischer and Triebenstein[1] examined a series of patients with senile and presenile cataract for evidences of tetany. They particularly examined the mechanical irritability of the ulnar nerve. This was done before and after examination for Trousseau's symptom. Among 68 patients 60 (88.2 per cent.) gave distinct symptoms of latent tetany. In only 11 per cent. were these absent. A control test was made of 12 patients of the same age. This showed that there were no signs of tetany in 10, present in one, and in one questionable. It should be stated that in Rostock tetany is not very unusual, and that quite a number of latent cases have been observed in the nerve clinic.

PITUITARY LESIONS[2]

In the hypophysis, just as in the thyroid gland, we can divide the symptoms into two groups according as they are caused by an over- or an under-production of hypophyseal activity. The symptoms which are produced by an **over-production** result in the condition known as gigantism, or as acromegaly, according to whether the condition occurs before the full skeletal growth or after. On the other hand, the conditions of **insufficient pituitary activity** give us the symptoms of dystrophia adiposo-genitalis (Froelich).

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[1] K. M. f. A., Vol. LII, p. 441.

[2] Henschen, Lewandowsky Handbuch d. Neurologie.
Cushing, The Pituitary Body, Lippincott, 1912.
Uhthoff, Graefe Saemisch-Hess, II ed.
Schueller, Lewandowsky Handbuch.
Schueller, Roentgendiagnostik, d. Gehirnkrankheiten in Allg. Chirurgie des Gehirnkr'k, Part II, 1914.

The hypophysis consists of a larger anterior or glandula lobe and a smaller posterior or nervous lobe bound together by a middle portion. The anterior lobe consists of cell columns which are surrounded by broad vascular sinuous spaces. The posterior lobe consists in a thin layer of cells which in places near the anterior lobe shows a thickening. An accumulation of these cells may lead to a cystic collection of colloid-like material. The pars anterior is a typical gland with internal secretion. Its colloid secretion is evacuated into the blood stream. In the pars nervosa the hyaline masses are the result of secretory activity. These are emptied into the liquor by way of the infundibulum.

The three parts of the hypophysis have different functions. The secretion of the anterior glandular lobe is connected with processes of growth in the skeleton and in connective tissue. An over-function of this lobe with the formation of adenoma leads in the growing individual to the unnatural development of the skeleton, in the full-grown to those curious connective-tissue thickenings suggestive of the picture of acromegaly. The posterior lobe consists of the nervous elements, the neuro-hypophysis, and its secretion is important for metabolism and the genital glands. In its injury the symptom-complex of dystrophia adiposo-genitalis results. Schäfer has shown that the middle portion, characterized by the presence of colloid, has an action upon the kidney vessels and kindey cells, and under certain conditions causes polyuria.

The anterior lobe is related to skeletal growth, while the posterior lobe furnishes hormones which act upon the heart, blood-vessels and kidney, the blood pressure, uterus, etc. It seems quite definite that acromegaly depends upon increased functional activity of the pituitary body and of its glandular anterior lobe, but it is not known whether the dystrophia adiposo-genitalis syndrome, characterized by general adiposity and genital atrophy, depends on an underaction of the entire gland or of its anterior portion or a dyspituitarism of the posterior lobe. The glandular insufficiency causes adiposity, high sugar tolerance, subnormal temperature, impotence, low blood pressure and great physical weakness.

While the anatomy of acromegaly is uniform, the pathological changes which lead to hypopituitarism are varying. The cause is most frequently tumors. These have the common features of exerting an injurious effect on the gland. The most frequent tumor is the adenoma. Then there is a group of epithelial tumors which are called infundibular tumors and are squamous epithelial growths. The third variety are a number of cystic tumors. Then there are neoplasms growing in the neighborhood, also aneurysms which produce similar changes. All these tumors resemble one another microscopically and in their topographic relationship. They frequently present cystic formations and areas of calcification. The size varies, usually from the size of a nut to that of a small orange. Sometimes a part of the tumor is situated within the normal or dilated sella; a second part extends along the base of the brain; and a thin pedicle often unites these two parts.

The symptoms of dystrophy are sometimes produced by hydrocephalic collections of fluid. These usually distend the third ventricle and compress the hypophysis. Similar conditions have resulted from circumscribed cyst-like accumulations of fluid in the cisterna of the chiasm and after circumscribed serous meningitis.

The **symptoms** may be divided, roughly, into four groups:

1. Local, or neighborhood symptoms.
2. General pressure symptoms.
3. Constitutional symptoms, those due to acromegaly or to the dystrophia adiposo-genitalis.
4. Correlation with other glands.

The most important neighborhood symptoms are **ocular** and are due to pressure on the chiasm. Ocular disturbances are present in **50 per cent.** of the cases. Now that our attention has been drawn to these cases, it is remarkable how many cases of hypopituitarism come to the oculist and how much more frequent eye symptoms seem to be in this condition than in acromegaly.

The ocular disturbances in hypophyseal disease depend upon the anatomic relationship of the optic chiasm to the hypophysis cerebri.

Normally the chiasm is not horizontal, but is inclined from above down and from behind forward. Its postero-inferior surface does not lie in the optic sulcus, but, as Testut has shown, it rests on the anterior part of the tent of the hypophysis, immediately anterior to the infundibulum.

The hypophysis is situated in the sella turcica, guarded in front and back by the clinoid processes, laterally by the cavernous sinus and its accompanying nerves, and above by the dural diaphragm which is perforated by the infundibulum, the connecting process between the pituitary body and the floor of the third ventricle.

The ocular symptoms are the result of direct pressure by the hypophyseal tumor on the basal visual paths and on the motor nerves of the eye.

As the pathological hypophysis grows in size it enlarges the sella in all directions, and as it extends upward it generally enlarges the infundibular hole and, like the tumors arising in the infundibulum, presses upon the ventral surface of the chiasm; the visual fibres which cross in the middle will first be pressed upon, producing characteristic disturbances. Within the crossed fibres in the center of the chiasm, and in the median line, are the crossed macular fibres. The visual disturbances then increase in an irregular way, and blindness may result. Characteristic visual changes may consequently only be present at a certain stage of the morbid process.

According to Henschen,[1] the typical course of the visual disturbances is as follows: Pressure on the ventral macular crossing fibres produces small macular or perimacular bitemporal scotomata upward; then pressure on the crossing ventral peripheric fibres results in bitemporal quadrant hemianopsia. Additional pressure then involves the uncrossed fibres, and one eye is blind with temporal hemianopsia in the other, or both eyes are blind. Sometimes the color fields are first involved, especially in the upper temporal quadrant. In general, the development of the field defect is irregular and not as stated above, and very different combinations of visual disturbances are observed.

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[1] Henschen, Lewandowsky, Handbuch, Vol. III, p. 751.

If the pressure is not exerted on the posterior surface of the chiasm, unusual combinations of visual disturbance may result from the enlargement of the pituitary tumor taking place anterior to the chiasm and then invading the orbit, from lateral pressure as Cushing[1] has observed, from an extension along one side of the chiasm, from an involvement of one or both optic tracts or of the optic nerves, and, finally, from a constriction of the optic tract or optic nerves by pressure against the anterior cerebral arteries.

The most frequently observed visual disturbances in pituitary disease are **defects in the visual field.**

Of these temporal hemianopsia occurs so often, in nearly 50 per cent. according to Uhthoff,[2] that it has been for many years regarded as one of the characteristic symptoms. This hemianopsia, however, is not symmetrical but is irregular with uneven limits, differing in this respect (de Lapersomne et Cantonnet)[3] from the homonymous hemianopsias. According to Cushing, the primary defect usually first involves the color boundaries alone in one upper temporal quadrant. This is followed by a more or less complete temporal hemiachromatopsia, possibly with a "slant" in the upper temporal form field, which gradually spreads downward until most of the temporal field is involved. The nasal field in turn shrinks away from the center as the blind field enlarges, though the process seems for a time to be arrested at the macular area. While one eye is blind there may be but little defect in the field of the other eye.

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[1] The Pituitary Body and its Disorders, Philadelphia, 1912.

[2] Graefe-Saemisch, 2d ed., Vol. XI, p. 1275.

[3] Manuel de Neurologie Oculaire, Paris, 1910, p. 308.

Cushing says it is of the greatest clinical significance to look for more tendencies toward temporal defects, particularly in the color peripheries. It must not be forgotten that bitemporal field defects are sometimes caused by a protrusion downward of the floor of the III ventricle in internal hydrocephalus.

Homonymous hemianopsia, according to Uhthoff, is very unusual. Cushing, on the other hand, finds that homonymous defects or tendencies in this direction are at least half as frequent as bitemporal ones. In 80 per cent. of Cushing's cases the field was involved bitemporally in 27, homonymous in 15; in 36 one or both eyes were blind, so that it was impossible to say which of the preceding forms had existed. Eight showed unusual or bizarre alterations. The typical hemianopsia, where the dividing line goes vertically through the point of fixation, is very unusual. The limiting line is usually irregular.

Scotomata, usually paracentral in location, have been described by a number of authors. They may precede the development of the temporal hemianopsia, or be observed on the disappearance of the hemianopsia after operation. It is difficult to explain their formation, unless by peripheric pressure on the chiasm just as in the case of the optic nerve. Nettleship[2] draws attention to central scotoma in hypophysis tumor as an early symptom which then goes on the temporal hemianopsia. The writer has observed a case of central scotoma associated with bitemporal hemiachromatopsia. De Schweinitz and Holloway[1] believe these scotomas are not uncommon, and suggest that more mention of them is not made because the visual fields were taken at a time when the scotomas were no longer present.

Concentric contraction of the field has been rarely observed. It is the expression of a peripheric optic-nerve affection.

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[1] Central Amblyopia as an Early Symptom in Tumor at the Chiasm, T. O. S., 97.

[2] Section on Ophth., A. M. A., 1912.

The course of the visual disturbance is usually slowly progressive, just as the growth of the tumor is slow. An intercurrent hemorrhage or œdema, or sudden increased size of a cyst, may cause an abrupt aggravation of the symptoms. On the other hand, the onset may be sudden and severe, with blindness, and then the condition partly clears up (Uhthoff). Daily variations in the field defects have been particularly noted by de Kleijn.[1]

Ophthalmoscopic Changes.—Simple optic atrophy is the most frequent, and was present in 20 per cent. of the cases collected by Uhthoff. Choked disc is only about one-half as frequent; this is explained by the tumor shutting off the entrance to the optic nerve sheaths. According to Cushing, a choked disc may become superimposed on the atrophic nerve head from the internal hydrocephalus complicating the pituitary tumor. Optic neuritis and neuritic atrophy are also only about half as frequent as optic atrophy. Melchior found atrophy in 27 cases, choked disc in 25, optic neuritis in 14. In addition to direct compression by the tumor, a number of observers have shown that the optic nerves are compressed indirectly through pressure exerted against the anterior cerebral arteries.

The ocular muscles are implicated in between 10 and 25 per cent., according to Uhthoff. These are nearly always oculomotor pareses, often in the form of ptosis. Affections of the other branches have been observed. Complete and bilateral oculomotor paralyses are uncommon. Paralysis of the abducens nerve is very unusual.

Nystagmus, changes in the pupillary reaction and exophthalmos have been observed in varying frequency; they are of no particular diagnostic importance.

X-Ray Examination.—Destruction of the sella varies as the growth is intrasellar or extrasellar. The intrasellar form produces a uniform dilatation of the sella. Its floor is depressed, thin and approaches the base of the middle cranial fossa. The dorsum is thin, elongated and pushed back. The anterior clinoid processes are normal or thickened or pushed forward. The extrasellar tumors produce a flattened pan-like distention of the entrance to the sella whereby the dorsum is thin and shortened, the anterior clinoids are shortened and sharpened and the floor which is thin does not seem to be nearer to the middle cranial fossa than normal.

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[1] Graefe's Archiv, Vol. LXXX, p. 307.

Erdheim has shown that tumors from the hypophysis itself, *i.e.*, intrasellar development, deepen the hypophyseal groove while the extrasellar developing tumors, usually arising from the hypophyseal ganglion, enlarge the entrance into the hypophyseal groove. The first are usually associated with acromegaly. The extrasellar tumors usually cause genital aplasia and obesity. Similar changes to the latter can also result from chronic hydrocephalus.

In *differential diagnosis* we must consider brain tumors situated in any part of the brain with secondary internal hydrocephalus. These may cause general cerebral symptoms and if the third ventricle be distended focal symptoms may be present of tumors in the hypophyseal region with X-ray deformities and the symptoms of glandular insufficiency. These cases can generally not be differentiated and are regarded as an under-group of the typus Froelich. Hypopituitarism has three characteristic symptoms:

1. Bitemporal hemianopsia.
2. Trophic disturbance: obesity, general atrophy.
3. Excavation of sella (X-ray).

The general symptoms of acromegaly in the typical cases are so characteristic that the diagnosis is easy, though there may be cases where the skeletal changes are slight and the presence of adiposity suggests the typus Froelich. In other words, a combination of hypo- and hyperpituitarism may exist just as we find a combination of the symptoms of exophthalmic goitre and of myxœdema.

IV POISONS[1]

Certain agents in toxic doses exercise a selective action on definite parts of the visual pathways. This permits a subdivision of the toxic amblyopias into the following groups: The first group presents the symptoms of a peripheral optic nerve lesion characterized by central scotoma and a free periphery such as occurs in the frequent alcohol and tobacco amblyopia. The second group shows changes which primarily are due to lesions of the retinal blood vessels, of which quinine amblyopia is the most striking example. Collins and Mayou[2] speak of another group in which the eye is affected secondarily to this effect of the poisons on the kidneys, as in lead poisoning and of agents such as salicylic acid and santonin which directly affect the cerebral visual centers.

Alcohol.—Of all the poisons which affect the eye, alcohol is probably the most frequent. The form of alcohol is usually the ethyl variety, though poisoning with methyl alcohol gives a very distinct and definite form of eye trouble. It seems that the stronger alcohols, such as brandy and whiskey, are the ones which exert a deleterious action on the eye, while beer or wines do not cause amblyopia. Furthermore, as Uhthoff states, the ones who are affected usually belong to the poorer, laboring classes where the indulgence in alcohol is apt to be excessive while the general nutrition of the body is below par. The male sex is very much more frequently affected than the female sex, though in East Germany Uhthoff found that women were affected in 10 per cent.

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[1] Uhthoff, Graefe-Saemisch, Vol. XI, 2, 1901.
de Schweinitz, Norris and Oliver, System, Vol. IV, 1900.
Lewin and Guillery, Die Wirkung v. Arzneimitteln und Giften auf das Auge, Berlin, Hirschwald, 1905.
Wilbrand und Saenger, Neurologie d. Auges, Vol. V, Bergmann, 1913.

[2] Collins and Mayou, Pathology, Philadelphia, Blakiston, 1911, p. 196.

Alcohol affects the eyes either acutely or chronically. Acute onset of the visual disturbance after excessive indulgence of alcohol is very unusual and is probably always due to the ingestion of methyl alcohol or products which are contaminated with this form of alcohol.

Ethyl Alcohol.—Chronic alcohol amblyopia is characteristic for this form of alcohol poisoning and is the frequent ocular change in alcoholism. Clinically the amblyopia which results from tobacco is identical and as these two agents are so frequently used together, the clinical picture is the same. It is now generally agreed that alcohol has more effect in bringing about this amblyopia than tobacco. At the same time either one of these two ingredients alone can produce typical toxic amblyopia. Uhthoff investigated 327 cases of toxic amblyopia and found that in these tobacco alone was the cause in 41. In the remaining 286 the cause was either alcohol alone or alcohol and tobacco combined. This toxic amblyopia occurs in patients beyond middle age. In other words, the minimum age is about 35 while under 20 the condition practically does not exist.

The visual disturbance is usually a gradual one, and the patients at first complain of their vision being fogged, the sight then becomes very much reduced. Uhthoff states that occasionally a curious improvement in vision takes place notwithstanding continued indulgence in alcohol and tobacco. An abstinence of alcohol and tobacco causes the symptoms promptly to disappear, while a return of the patient to his old habits is followed by a prompt deterioration of vision. The course is a distinctly chronic one, lasting for weeks and months, often for years. The prognosis, as to blindness, is favorable; complete recovery is possible if the amblyopia has not advanced too far and the ophthalmoscopic changes in the optic nerve are not permanent (temporal atrophy). The condition does not go on to total optic atrophy and blindness; the cases reported in literature with this outcome Uhthoff believes to have been incorrectly observed and a complication overlooked.

The change in the visual field in this form of toxic amblyopia is a typical one. It is characterized by the periphery of the field remaining unaffected, while a central scotoma is present. This scotoma is generally relative. Another characteristic is that the lesion is always bilateral. The usual form of scotoma is a horizontal oval which extends temporally from the point of fixation. In some cases there are pericentral scotomas which extend from the point of fixation in any direction. Uhthoff regards as very characteristic the observation, first made by Samelsohn, which consists in the gradual diminution of the functional disturbance from the center to the periphery of the scotoma so that while in the center of the scotoma there is an absolute defect, in the periphery of the scotoma the defect is only relative or the defect in the center will be for red, green and blue, while in the periphery the blue will be recognized. The point of fixation is not generally the point where the greatest functional disturbance takes place, but this is situated somwhat excentrically, a spot which has been called by Sachs "Kernstelle." Then as the process improves this is the part in which the functional defect disappears last. The peripheric limits of the scotoma vary. In marked cases there may be no appreciation for red and green. A peripheric contraction of the field, in addition to a central scotoma, is a very unusual symptom in Uhthoff's experience, and if this persists, there are probably two different anatomical factors present.

The ophthalmoscopic changes consist in a partial atrophic discoloration of the temporal parts of the disc in over one-half of the cases (Uhthoff). The nasal half may show a certain degree of pallor, though the contrast between the two is always present. This discoloration of the temporal half in toxic amblyopia does not occur short of some weeks or months. There are cases in which it has been absent for a long time. Second, in frequency, the ophthalmoscopic changes consist in hyperemia and slight discoloration of the disc (8 per cent. Uhthoff). This is usually observed only in recent visual disturbances, though as a rule in these recent cases the ophthalmoscopic condition is negative.

The pathology of this condition has been studied by a number of authors and is regarded by one group as an interstitial neuritis, while the second, basing their views upon the examination of the retinal elements by recent methods of staining, as a degeneration of the retinal ganglion cells. Experimental investigations of Holden and Birch-Hirschfeld have shown pathological changes in the ganglion cells of the retina and in the optic nerve. Collins and Mayou[1] regard the retinal changes as primary and the atrophy of the optic nerve as secondary, the leucocytosis into the nerve serving to remove the débris of the broken down medullary sheath; whileother authors believe the leucocytosis to be a part of a retrobulbar neuritis and the changes in the ganglion cells consequently as secondary. Uhthoff found pronounced pathological changes in the optic nerves more frequent (14 per cent.) in cases of severe alcoholism than in peripheric multiple neuritis (4 per cent.).

Alcohol amblyopia is frequently complicated with peripheric multiple neuritis. Thus Rennert and Myles Standish[2] found partial atrophic discoloration of the discs in 5 to 6 per cent. of alcohol neuritis cases, and slight neuritic changes in the discs without visual disturbances were present in other cases.

Nicotine.—The harmful effect of tobacco is probably due to nicotine or to one of its derivatives, pyridine, which is contained in varying quantities in the various forms of tobacco.

The form in which tobacco is used varies in its toxicity. Cigarettes are probably the least harmful, the smoking of strong tobacco in pipes and strong cigars are usually the toxic conveyors. Amblyopia has even been reported after taking snuff and "dipping," and de Schweinitz mentions its occurrence in those who work in tobacco factories. Certain nations, Spaniards and Turks, seem to enjoy a relative immunity to the injurious effects of tobacco. Tobacco causes a chronic amblyopia and cases of total atrophy with amaurosis after nicotine poisoning are questionable, and Uhthoff believes that in these cases just as in the cases which occur after alcoholism, the complicating optic nerve processes of either spinal or cerebral origin have been overlooked. The symptoms of alcohol and tobacco amblyopia cover one another, so that it is not necessary to repeat them here. At the same time, tobacco alone can produce disturbances of vision, though this has been questioned by some authors. The time at which the tobacco affects the optic nerve corresponds to what has been said about alcohol. In the experience of one observer the tobacco amblyopia occurred only in individuals who began to smoke before the twenty-first year and then usually occurred between the fortieth and forty-fifth year. It is evident that those in good bodily vigor are less susceptible and disturbances of the general health, particularly of a gastric nature, seem to be an important predisposing factor.

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[1] Collins and Mayou, Pathology and Bacteriology, p. 197, Blakiston, Philadelphia, 1911.

[2] Quoted from Uhthoff, *l.c.*

Collins and Mayou[1] state that vascular sclerosis plays an important rôle in nicotine amblyopia and if the vascular sclerosis be well marked, recovery notwithstanding the discontinuance of tobacco may be delayed or not occur at all. Visual disturbances have been noted in horses after poisoning with a plant known as Australian tobacco.

Methyl (wood) alcohol causes a sudden loss of eye sight accompanied with symptoms of an acute gastro-intestinal disorder. Wood alcohol is usually taken in place of the ordinary grain alcohol for which it serves as an adulterant in cheap whiskey, cordials, Jamaica ginger, bay rum, etc. It is inhaled in the process of shellacking beer vats, and var-

[1] Pathology, p. 199, *l. c.*

nishing pianos. Wood has reported a case where wood alcohol was applied externally to the skin for the relief of muscular pains and caused toxic symptoms and blindness. After drinking wood alcohol the patient suffers from abdominal pain, prostration, headache, nausea and vomiting; the symptoms may increase and lead to death or recovery with blindness. The loss of sight is rapid and complete, a slight improvement after a few days is common, but sight is again lost. The ophthalmoscope shows slight neuritic changes and later optic atrophy. Some observers have reported on extensive retinal œdema especially along the retinal vessels. Fridenberg[1] has drawn attention to a peculiar change which the atrophic disc undergoes in the late stages following wood-alcohol poisoning, consisting in a deep excavation which resembles a glaucomatous cup of a pure white color. The central vessels show signs of previous neuritis, and there are atrophic changes and pigment about the nerve head in the retina.

The pupils are dilated and immobile, sometimes movement of the eyeballs is painful. In the course of the disease an early though generally only temporary improvement is frequently observed. The prognosis is poor, though some cases of restoration of vision are on record. The inhalation cases are usually milder and recovery occurs.

Bisulphide of Carbon.—The use of this drug in rubber manufacture has led to cases of general intoxication both in acute and chronic forms. The latter is the more frequent, and is generally the one in which the eye is involved. The onset of the visual disturbance is a gradual one, though exceptionally it begins acutely. The patients complain of seeing through a cloud; there is the characteristic central disturbance of vision with a comparatively free periphery of the field and the loss of color perception in the center. The condition is usually bilateral and all the reports agree on the presence of the central defect with normal periphery of the field, characteristic for toxic amblyopia in general.

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[1] Fridenberg, T. Am. O. S., 1910.

The ophthalmoscopic changes consist in a slight optic neuritis followed by partial atrophic discoloration, though the fundus picture is often negative. On the whole, the similarity of the carbon bisulphide amblyopia with that of tobacco and alcohol is unmistakable, except that the visual disturbance in the former is more marked, developing more rapidly with more pronounced ophthalmoscopic changes. The cases with visual disturbances show symptoms of general poisoning, such as weakness of the extremities, sensory disturbances, headache, vomiting and vertigo. A multiple peripheric neuritis may be a co-existing condition, though this is unusual and has been reported as generally occurring without visual disturbance.

Iodoform.—At the time in which iodoform was a universal remedy in the antiseptic treatment of wounds, cases of general intoxication occurred. In some of these visual disturbances resulted in the form of optic neuritis with the characteristics of a toxic amblyopia with central scotoma and a preserved peripheric visual field. This later on was followed by a temporal atrophy of the optic nerve.

Stramonium.—Stramonium cigarettes are used in asthmatic attacks. After the use of these cigarettes amblyopia has resulted which gave the usual toxic symptoms similar to those found in tobacco and alcohol.

Thyroidine.—The treatment with thyroid products, which has been very much more general in recent years, has led to visual disturbances with symptoms of a retrobulbar neuritis and the presence of a central scotoma. According to Coppez, the optic discs show greater changes than in the usual toxic cases. They are hyperemic, the veins are dilated and tortuous. The ocular disturbance does not occur until after the remedy has been used for a long time and it is associated with disturbances of the general health. The prognosis is good and improvement has followed the cessation of the remedy.

Quinine.—Quinine causes visual disturbances by affecting the optic nerve and the retina in a way which is characteristic and differs from any of the agents just described. While the dose is usually a large one, Collins and Mayou state the smallest dose recorded as causing defect of sight is 5 grams given in 38 hours, and de Schweinitz has seen 12 grains produce decided temporary amblyopia in a susceptible and neurotic woman. Quinine amblyopia has been observed in sufficient numbers to demonstrate distinct characteristics and a uniform clinical picture. The form of development, the course, the visual fields and the ophthalmoscopic changes have all contributed to the establishment of a particular form of amblyopia, as was first described by Gruening and Herman Knapp. The size of the dose of quinine which produces the amblyopia varies. A certain predisposition or idiosyncrasy is present in some of the cases and cachectic and weakened general conditions facilitate the onset of the amblyopia. In mild cases the visual disturbances are transient and are not severe. A large dose is usually followed by complete blindness which is always accompanied with other symptoms of poisoning, particularly with disturbance of hearing. This blindness continues for a variable length of time and then gradually disappears after a period of hours, or days, or sometimes weeks. The central vision is the first to return, while the peripheric vision often remains defective in the form of a concentric contraction of the field.

Quinine amaurosis is not permanent, but marked loss of sight may persist. The visual disturbance is always bilateral. The change in the field consists in a concentric contraction of varying degree. This is accompanied with a diminution of central vision, though the latter frequently is regained to a remarkable extent.

An interesting and important feature of this amblyopia is the ophthalmoscopic condition. The most striking change is a change in the vessels. When the case is observed early there are symptoms of ischemia of the retina;

there is diffuse white clouding of the retina in the region of the posterior pole, a cherry red spot at the fovea and the margins of the disc are often blurred. In a few days the contraction of the retinal vessels and a pallor of the disc are distinct. The contraction of the vessels can occur to such a degree that they appear almost obliterated. The cause for this amblyopia consists in circulatory changes in the retina and in the optic nerve and the visual disturbance is explained by the ischemic changes in the retina. In addition to this action upon the blood vessels some authors believe that quinine exerts a paralyzing effect upon the light perceiving elements in the retina. Uhthoff regards it as doubtful that the contraction of the retinal vessels is due to a direct vascular spasm and believes that the reduction in blood pressure and the involvement of the cardiac action are probably responsible. In the later stages distinct anatomic changes develop in the vessels, such as thickening of the walls, endovascular processes, obliteration and thrombosis. In the early stage of the visual disturbances Holden and others have drawn attention to the absence of organic changes in the vessels. They regard the ischemia as a predisposing element to which vascular changes are subsequently added. Some authors (Holden, Druault), based on an examination with recent staining methods, have found a degeneration of the ganglion cells of the retina. These changes in the ganglion cells are preceded by a curious albuminous serous exudation into the nerve fibre layers. Holden believes that these retinal changes are probably due to a lack of circulation.

Ethylhydrocuprein (**optochin**), a quinine derivative and a specific in pneumococcus infections, when given internally in pneumonia has caused toxic amblyopia. Fränkel[1] observed amblyopia in 14.3 per cent. The amblyopia disappeared promptly on stopping the drug. Oliver[2] observed tinnitus, deafness and loss of sight. There was light perception only and dilated pupils. The retinal vessels were reduced to fine threads; the optic nerves were white. There was partial recovery with concentrically contracted fields. Weeks[1] reported a case of pneumonia treated with optochin at the Rockefeller Institute, which developed a typical quinine amblyopia; final vision was $^{20}/_{30}$, and a moderately contracted field.

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[1] Fränkel, B. kl. W., 1912, p. 664.

[2] Oliver, B. M. J., Apr. 22, 1916.

Nitrobenzol.—Nitrobenzol causes symptoms of poisoning in those working in anilin factories. The dinitrobenzol is used in factories which manufacture explosives. Both of these agents cause general symptoms of intoxication, cyanosis, jaundice, headache, prostration, changes in the blood, etc. They furthermore produce a disturbance of vision which occurs in the form of a peripheric optic nerve lesion. The symptoms consist in a concentric contraction of the field, a central scotoma with changes in the eye grounds and venous hyperemia followed by a pallor of the discs. The changes in the blood resemble those found in pernicious anemia according to Uhthoff, and may be of moment in causing the ocular changes. The pupils are affected in proportion to the severity of the general disturbance and are usually dilated and immobile.

Anilin.—Anilin resembles nitrobenzol in the similarity of the general symptoms of poisoning. The disturbances of vision are also the same. The agent is probably introduced through the inhalation of fumes. Changes in the conjunctiva and in the cornea have been observed from the direct action of these fumes. A number of cases of direct injury of the conjunctiva[2] and of the eye ball with anilin pencils has been observed. The symptoms, aside from the discoloration of the tissue which often lasts for a long time, consist in a very severe inflammation of the eye itself. Deep corneal ulcers have led to perforation and the formation of corneal staphyloma. Anilin is said to cause changes in the retinal vessels, and retinal hemorrhages have also been observed.[1]

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[1] Weeks, Archives of Ophthalmology, 1916.

[2] Mellinghoff, Kl. M. f. A., 1906, Vol. XLIV.

The **aryolarsenates** which comprise atoxyl, soamin and arsacetin, are compounds of anilin and arsenic; and as Collins and Mayou[2] state, arsenical poisoning has not caused loss of sight, the cases of blindness which have followed the administration of these drugs in syphilis and sleeping sickness must be due to the anilin constituent. They cause progressive optic atrophy without inflammatory changes and end in blindness.

Morax found that the loss of sight is rapid, with contraction of field or blindness and optic atrophy; the loss is generally stationary but partial recovery has been observed. The toxic dose is variable owing to idiosyncrasy, hence its danger.

Sattler found in arsacetin that the toxic agent attacks the ganglion cells and the anterior part of the optic nerve, sparing the papillo-macular bundle.

Atoxyl causes retrobulbar neuritis, atrophy, contracted arteries, concentric contracted fields and blindness. It attacks various parts of the optic paths, retina, optic nerve or optic ganglia. The toxic action is due to the chemical combination and not to either constituent, while others regard the anilin as the poisonous agent.

Schirmer observed a case of atoxyl poisoning where the vision was gradually lost, after the fields showed nasal peripheric contraction and no scotoma; the optic nerves were pale with narrow arteries. Atoxyl, if it produces visual disturbances, usually leads to blindness (Birch-Hirschfeld) from primary nerve degeneration.

Filix Mas.—Another agent which produces symptoms somewhat like quinine is filix mas. The active principle is filix acid and its toxic action is influenced not only by the individual predisposition, the cachectic condition of the patient, but also the method in which it is administered, for when accompanied with castor oil it seems to be much more toxic than otherwise. The toxic dose varies. According to Bravitz[1] a dose from 8 to 10 grains is never to be increased. In some statistics the poisoning with filix mas has been unusually severe. Siedler-Huguenin mentions 78 cases of whom 12 died and 33 suffered from bilateral blindness, while the blindness in 15 was one-sided. The reports on the changes in the visual field are not definite, but the changes do not seem to be those of a retrobulbar neuritis (central scotoma with free periphery), and in most cases the entire field was affected. The general symptoms have been so severe and the loss of sight so rapid that an examination of the field was generally not possible. In a part of the cases the visual disturbance is one-sided which is a proof of the peripheric nature of the disease. The ophthalmoscopic changes, if the visual disturbance has lasted for some time, consist in atrophy of the optic discs with a sharp margin. In many cases there are changes in the retinal vessels. Siedler-Huguenin believes that filix acid exerts a direct poisonous toxic action upon the retinal vessels.

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[1] Chiari and Berger, Danger of Anilin in Cosmetics. Arch. d'ophth., XXX, p. 706.

[2] *l.c.*, p. 203.

The similarity between these visual changes and those found in quinine have been noted by a number of observers; at the same time the prognosis in filix mas is surely very much worse than in quinine. The general symptoms of intoxication of the two drugs are also similar (loss of hearing, vertigo, stupor, tremor, convulsions, headache, vomiting, etc.).

Lead.—In lead poisoning the eye may be involved in a variety of ways. There may be a disturbance of vision or of an ocular muscle. Lead amblyopia, however, is an unusual condition. Groenouw speaks of it occurring in about 1 per cent. in the cases of lead poisoning. The visual disturbances are characterized by being manifold. On the one hand, there is a direct affection of the peripheric optic paths. On the other hand, there are changes which result from primary disease of the blood vessels and, third, cerebral changes occur which produce secondary disturbances of vision. Finally, as a result of lead poisoning, changes in certain organs (chronic nephritis) result which may secondarily involve the sight.

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[1] Quoted from Uhthoff.

The visual disturbances may be grouped as follows: I. A bilateral more or less complete blindness of rapid occurrence which usually improves though leading in a small proportion of the cases to permanent changes. The ophthalmoscopic condition is negative. These changes have been observed in attacks of lead colic and in connection with epileptic attacks. II. Very much more frequent is lead amblyopia with the characteristics of an affection of the peripheric optic nerves, which does not cause complete blindness. The beginning is generally gradual though in one-fifth of the cases it was rapid, sometimes momentary. The ophthalmoscopic changes are optic neuritis with hyperemia, choked disc, neuroretinitis, or neuritic optic atrophy, particularly in the temporal quadrant. In 10 per cent. of the cases there is no ophthalmoscopic change and the change in the visual field suggests a localization of the process in the optic nerve. Changes in the retinal blood vessels have been noted in many of the reports. These consist in changes in the walls, a constriction, perivasculitis, white sheathing, endarteritis, or vascular spasm. In addition to these vascular changes hemorrhages and white plaques have been observed in the retina.

The examination of the field has shown in a number of cases a central scotoma with a preserved periphery, though this is very unusual. Lead amblyopia is usually much more acute and more pronounced than the toxic amblyopia from alcohol and tobacco. The more prevalent change in the field consists in a uniform peripheric contraction or a sector-like defect. Homonymous hemianopsia has been observed in a number of cases. The visual disturbances following lead poisoning are always bilateral. The prognosis varies, and the cases in which the loss of sight is rapid offer the best prognosis.

The ocular muscles are affected in not a small percentage of cases. The abducens leads in frequency and in general both nerves are affected. Next in frequency is the oculomotor nerve which may include the internal muscles; combined paralyses of the ocular muscles have also been observed.

Lead intoxication with its resulting ocular disturbances is usually caused by occupational diseases. The patients are generally workmen in lead factories, painters, typesetters, printers, workers in paper factories. The use of certain remedies such as hair dyes, powders, plasters, have been known to produce toxic symptoms. The patients with ocular disturbances always show other symptoms of lead poisoning: lead colic, pain in the joints and in the head, epilepsy, paralysis, blue lines in the gums, anæmia, nephritis, etc.

Salicylic Acid.—Visual disturbances after salicylic acid or its substitutes have a distinct similarity to those produced by quinine; and the general symptoms of poisoning, particularly those of hearing, present analogies to quinine poisoning. On the whole, salicylic acid causes visual disturbances much less frequently and less intensively. The visual disturbances have generally occurred without ophthalmoscopic changes. Exceptionally pallor of the discs with contraction of the retinal vessels have been mentioned. The prognosis is favorable as in all cases vision has been restored. The changes in the visual field are not characteristic. It is perhaps to be noted that changes in the pupil, generally a dilatation, sometimes contraction or iridoplegia, have been observed. Whenever the pupils have been found dilated, symptoms of severe general poisoning were present.

Arsenic.—Ocular changes due to poisoning with arsenic are very unusual. Œdema of the eye lids and conjunctivitis are probably the most frequent. An affection of the optic nerve in the form of optic neuritis has been reported. It is generally mild and gives the functional symptoms of a toxic amblyopia.

Schirmer[1] did not succeed in finding more than 6 cases of optic nerve affections after inorganic arsenic preparations and these did not show the same features as the atoxyl amblyopia. The clinical features are only a moderate impairment of vision, the visual field is normal except for a central color scotoma. The disc is not sharply outlined, the arteries are narrow, and later there is temporal pallor. Prognosis is good; recovery occurs on discontinuing the drug. There is not a single case of blindness on record.

Silver Nitrate.—Though toxic effects of silver just as lead may show themselves in a paralysis of the radial nerve, gout and albuminuria, but very few cases of ocular disturbances secondary to this condition have been observed. A peripheric disturbance of the optic nerve has been reported following the prolonged use of this chemical in a hair dye. The symptoms were those of a toxic amblyopia with central scotoma.

Phosphorus.—In poisoning with phosphorus the eye is generally not affected. Uhthoff mentions as the most frequent symptom the discoloration of the conjunctiva observed in general jaundice. Changes in the fundus have been described in the form of hemorrhages of the retina.

Antipyrin.—Antipyrin has caused inflammatory changes in the conjunctiva and of the lids. These symptoms were associated with swelling of the face, skin eruptions, hoarseness and coryza.

Coal-gas Poisoning.—The disturbances of vision which have been observed after coal-gas poisoning seem to have been central in origin, often accompanied with cerebral lesions. The ophthalmoscopic changes were venous hyperemia. In addition to the visual disturbances, there have been some unquestioned cases of paralysis of the ocular muscles. Uhthoff is inclined to regard these as due to hemorrhagic changes in the nuclei.

[1] Schirmer, Arch. of Ophth., Vol. XXXIX, p. 464, 1910.

Mydriatics.—In addition to the physiological action of atropin, this drug not infrequently causes distinct symptoms of poisoning. Aside from its action in developing glaucoma in eyes which are predisposed to this disease, its prolonged use in the form of eye drops is often followed by a follicular conjunctivitis. The cause for this is sometimes ascribed to contaminated eye drops, though in many cases this is not the case. In addition to this action on the conjunctiva in some people the instillation of atropin causes a swelling and redness of the eye lids with marked itching. These changes are so pronounced that they resemble an attack of erysipelas. They occur particularly in older people with rheumatic tendencies and are regarded as the expression of an individual idiosyncrasy. The general symptoms of atropin poisoning are particularly prevalent in children in whom they cause restlessness, dryness of the throat, excitement and even delirium. In Uhthoff's monograph the frequency of visual hallucinations is mentioned. A number of cases of mental disturbance following the use of atropin have been described. The delirium which is sometimes observed after cataract operations in old people may possibly be partly due to atropin instillations. As a substitute for atropin, scopolamin is not infrequently used. This, in the writer's experience, however, has been more apt to produce general symptoms of poisoning in old people. The patients have complained of a numbness and weakness in their limbs and have had difficulty in speaking and showed evidences of distinct mental aberration.

Chloral Hydrate.—Chloral hydrate causes contraction of the pupil and diplopia; visual hallucinations have, in addition, been described. After prolonged use, conjunctivitis and swelling of the lids were observed.

Pellagra.—Pellagra is a chronic and periodically recurring intoxication from contaminated grain which occurs endemically in certain tropical countries. Michel speaks of an erythema extending from the skin of the cheek to the eyelids, often accompanied with vesicles and blebs. When the swelling has disappeared the areas affected are darkly pigmented. The disease begins in the spring months and is accompanied with general symptoms of prostration. The desquamation continues until the winter and in the following spring the symptoms are renewed. The optic nerve is affected in the form of an optic atrophy which may be accompanied with blindness. It is difficult to define the exact nature of this nerve affection, whether it is a simple degeneration or an optic neuritis.

There are a number of reports of inflammatory changes in the disc and in the retina. These consist in hyperemia of the optic nerve heads, tortuous retinal vessels, anemic discs with thin blood vessels. Hemeralopia is a symptom which has been dwelt upon by a number of authors which occurs partly in the form of idiopathic night blindness without ophthalmoscopic changes, in others in conjunction with pigment degeneration of the retina.

Welton[1] states that there are no eye symptoms characteristic of pellagra and that they are more the expression of general weakness and are proportional to the severity of the disease.

Ergotism.—Poisoning with ergot can produce a variety of ocular lesions. There are no records of distinct involvement of the optic nerve, though some authors have reported upon ophthalmoscopic changes, consisting in an anemia of the retina with contraction of the retinal vessels. Slight and transient visual disturbances have been reported. The occasional development of cataract seems to be definite; it is always bilateral and can occur at any time of life. It is particularly that form of ergotism which is associated with attacks of convulsions in which the development of cataract seems to occur.

Botulism.—Meat can at times act as a poison. Thus, certain forms of meat, like fish, may act as a poison or the meat may be diseased or decayed. It causes either symptoms of paralysis or poisoning with ptomaines or, finally, acute gastro-enteritis. The decay of the meat need not necessarily be observable and the meat may taste and appear fresh. Numerous observations on ocular disturbances after poisoning with meat, fish or sausage have emphasized the prevalence of a disturbance of the inner muscles of the eye, namely, the sphincter of the pupil and the muscle of accommodation. Next in frequency is an involvement of the external muscles, particularly of ptosis. Both the sphincter of the pupil and the ciliary muscle are affected simultaneously. Occasionally the pupillary reaction to light is less affected than that of accommodation. This is in striking contradiction to the condition found after diphtheria.

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[1] Welton, J. A. M. A., Nov. 13, 1909.

The external muscles most frequently involved are those of the elevator of the upper lid and this is an isolated symptom, together with the internal ophthalmoplegia. In one-quarter of the cases other external muscles supplied by the III, IV and VI nerves have been affected. It is very unusual to find a lesion limited to an external muscle with the picture of an ophthalmoplegia externa while the inner muscles are intact.

The prognosis is good and recovery takes place after a certain length of time. The onset of the visual disturbance is rarely early after the poisoning, and is generally after an interval of from three to ten days. The similarity of these symptoms to those found in atropin poisoning has been commented upon and Leber (quoted from Uhthoff) has drawn attention to changes in the function of certain secretory nerves (saliva, sweat, and tears). At the same time the frequent associated involvement of the external muscles in botulism and particularly the involvement of the accommodation, should be emphasized. The ocular disturbances are always bilateral. It is questionable whether a true amblyopia due to changes in the optic nerve or in the retina occur. Definite ophthalmoscopic and anatomic changes have not been found. The agent which causes the poisoning is usually due to the ingestion of sausages, certain forms of meat, ham, veal, goose, etc., oysters, crabs, mussels, sour cheese, and milk.

V. INFECTIOUS DISEASES

TYPHOID FEVER

A variety of affections of the eye have been reported after typhoid fever. These comprise corneal ulcer, iritis, metastatic choroiditis and optic neuritis. Uhthoff found 17 cases of optic neuritis in his statistics of 253. Groenouw collected 20 cases from the literature. Visual disturbance occurred in both eyes in the second or third week, though sometimes somewhat later. The visual field showed a number of variations and a central scotoma has been observed. The ophthalmoscopic picture was that of an optic neuritis or of neuroretinitis. The outcome is more or less complete recovery. The optic neuritis is regarded by some authors as a part of an inflammation of the brain or of its membranes, evidenced by the presence of severe cerebral symptoms, paralysis of the ocular muscles, hemiparesis, etc. The paralyses of the ocular muscles occurring in typhoid fever are also an indication of a cerebral complication. Some of these persist for a number of weeks or for months, while in other cases the condition was permanent. The tendency to hemorrhages which is present in typhoid may show itself by hemorrhages in the retina or in the skin, conjunctiva or orbit.

SMALL-POX

The eyes were frequently involved in small-pox before the general introduction of vaccination. At the present time ocular complications are very unusual, though perhaps not entirely unknown. Groenouw states that before vaccination 35 per cent. of all blind lost their sight from small-pox. According to Adler, mentioned by Groenouw, the ocular lesions are hyperemia of the conjunctiva, pustules on the lid, conjunctiva and the lacrymal passages. During the subsequent stage severe diseases of the cornea, iris and retina and of the entire eyeball are noted. In the skin of the eyelids the small-pox papules appear very frequently together with œdema of the lid. This results usually in the patient being blind for a number of days. A secondary infection is erysipelas or abscesses of the eye lids and gangrene of the skin. The pustules in the intermarginal spaces have an entirely different appearance. The conjunctiva is regularly involved. It is first hyperemic, the secretion is somewhat increased, and pustules may appear on the conjunctiva just as well as on any other mucous membrane of the body. The corneal affection is usually a secondary one, as true pustules have not been definitely found present.

Roche[1] concludes that the ocular complications are almost exclusively a late keratitis due to secondary infection, and that they could be favorably influenced by the proper preventive treatment.

Corneal abscesses or very severe ulcers are the sequelæ of the corneal processes; they may be followed by a number of severe complications on the part of the eye. Iritis is also a complication which depends upon a general infection of the body. Adler found it present in 13 per cent. of the cases. It usually occurs in the stage of desquamation or in convalescence. A number of diseases of the eye ground have been observed; choroiditis, retinitis and optic atrophy. The lacrymal apparatus and the bones of the orbit have also been involved.

CHICKEN-POX

Unusually severe eye complications have been observed in chicken-pox, though these are probably all due to a secondary infection. The skin of the lids is usually the seat of an eruption of small vesicles. Gangrene of the lid has been a sequel. The vesicles involve the conjunctiva just as they affect the mucous membrane of the mouth and pharynx. Iritis and optic neuritis have been reported.

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[1] Roche, Annales d'oc., Vol. CXLIV.

VACCINIA

A patient that has been vaccinated sometime suffers from an infection of the eye with the vaccine. As a rule the ones affected are children who have been vaccinated or women who are taking care of vaccinated children. The infection takes place through the finger by which the infectious material is directly inoculated in the eye lids. According to Schirmer, out of 47 cases, the lids were affected in 43, the conjunctiva in 3 and the cornea in 1. In the lids the vaccinia occurs in the intermarginal space, particularly the angles of the lids. At the lid margin the vesicles are different than the ones observed in the skin and usually appear as flat ulcers covered with a membrane. There is moderate pain and the lids are swollen. The œdema is a characteristic symptom. The neighboring glands are involved and painful. There may be an associated conjunctivitis with a great deal of discharge. The ulcer during the next few days enlarges or new ulcers may appear, and not infrequently secondary ulcers will appear, exactly at the contiguous part of the opposite lid. The symptoms disappear after eight to twelve days and leave practically no scar. The mild course of these apparently severe ulcers is due to the superficial character of the process. Primary vaccine vesicles on the conjunctiva have been occasionally observed, though as a rule the conjunctiva is secondarily affected. The vaccinia in this location appears like the ulcer just described. The cornea is usually involved secondarily to the lid vaccinia and two forms of keratitis have been described. In the first there is a peripheric infiltration and an ulcer; in the second there is a deep keratitis. The corneal ulcer may appear in any stage of the disease; it is not characteristic and usually heals without complications. The keratitis profunda, appears later in the course of the disease, after the swelling of the lids has disappeared, and consists in a gray opacity situated in the deeper layers of the cornea. Iritis is always present and there may be corneal deposits. The disease is obstinate and may lead to permanent clouding of the cornea. This peculiar form of keratitis shows some similarity to disciform keratitis. Schirmer believes it to be an ectogenic infection from the surface of the cornea in which the superficial ulcer heals.

SCARLET FEVER

While conjunctivitis is characteristic for measles, it is usually absent in scarlet fever. There may be a slight reddening of the conjunctiva with increased secretion during the stage of eruption. Later, during the desquamation a severe conjunctivitis is sometimes present. Diphtheritic conjunctivitis is a very serious condition which occurs in the so-called scarlatinal diphtheria and is a streptococcus infection.

The various diseases which follow scarlet fever are not in any direct relationship to the disease itself, but are a continuation of preëxisting conditions or in which this infection has been the exciting cause. Uhthoff found optic neuritis in only 3 cases among 253. In 5 cases which Groenouw collected, albuminuria was present in 1, while in 3 the urine was free from albumen. The margins of the discs were blurred, the swelling sometimes was sufficient to form a choked disc, the surrounding retina was changed and hemorrhages were present. Orbital abscesses have been observed in a number of cases and the lacrymal apparatus has also been involved. The ocular muscles are rarely affected; a paresis of accommodation has been observed in a few cases.

The eye is sometimes indirectly affected in scarlet fever. Thus, the nephritis of scarlet fever often shows itself first by an œdema of the eye lids which may be more marked on one side than on the other. Another symptom is uremia, which may lead to amaurosis. Scarlatinal nephritis is one of the most frequent causes for this form of blindness. The amaurosis is observed in the stage of desquamation in the third to fourth week after the eruption. Albuminuria is always present, though during the attack of uremia it may be absent. Blindness is bilateral and absolute. Its onset is usually sudden and is generally accompanied with severe cerebral symptoms; headache, vomiting, convulsions and stupor. The blindness lasts for two to three days. If the patient does not die, sight is restored. Albuminuric retinitis in scarlatinal nephritis is unusual. The eye may also be affected secondarily to a purulent otitis, which is so frequent a complication of scarlet fever.

MEASLES

In measles *conjunctival catarrh* is one of the typical initial symptoms. The patient has fever and catarrh of the entire respiratory tract, which includes the conjunctiva. The conjunctiva of the eye lids is reddened and there is distinct discharge; in some cases the congestion may include the ocular conjunctiva. An unpleasant sense of pressure and photophobia are the subjective symptoms. The height of the inflammation occurs usually on the second day after the beginning of the eruption. This exanthem usually begins in the mucous membrane of the gums and it is immediately followed by that of the conjunctiva. Monti regards the reddening and swelling of the retro-tarsal folds as characteristic for measles, while the palpebral conjunctiva is at first unaffected. Pale, irregular spots have been noted on the conjunctiva, particularly along the free margin, more rarely at the fornix. The number of these is in direct proportion to the involvement of the skin. The conjunctival eruption disappears rapidly. The conjunctiva becomes uniformly red and the conjunctivitis rapidly runs its course, assuming its normal color at the time of the onset of the desquamation. The catarrh of the conjunctiva like that of other mucous membranes is produced by the specific agent of measles, because the disease has been transmitted by the secretion of the conjunctiva. Other complications are probably due to infection with other organisms. This applies to the severe forms of conjunctivitis, keratitis and metastatic diseases of the eye. Blepharitis, eczema of the lids and hordeola are frequent sequels. Hordeola occur as ordinary styes or as a suppuration of a Meibomian gland, the so-called internal hordeolum. In measles the conjunctivitis sometimes assumes a membranous character which in turn may lead to corneal involvement. Eczematous lesions of the skin of the face and of the nose are frequent, particularly in children in poor general health. A frequent and important sequelæ is phlyctenular disease of the eye. This occurs after the acute symptoms have passed.

Aside from the usual conjunctivitis, the eye is rarely affected in measles. On the other hand, very severe lesions may occur. These are generally in young children between two and three years of age, and who are in very poor general condition, with broken-down glands. A number of cases of metastatic ophthalmia have been reported. Inflammation of the optic nerve is very unusual; Uhthoff in his statistics found only 9 cases out of 253. Groenouw has found 17 cases reported in literature. Most of these cases were in children under ten years of age. Diseases of the lacrymal organs and affections of the orbit have also been described. Paralysis of the ocular muscles after measles are in part due to a peripheric neuritis and in others to a circumscribed encephalitic focus in the brain or to meningitis. Paralysis of the accommodation is very unusual.

WHOOPING COUGH

Hemorrhages into the conjunctiva and eye lids are a frequent occurrence in whooping cough. They are regarded as the result of venous congestion and disappear after a number of weeks, without leaving any trace. Hemorrhages have also been observed in the orbit, in the anterior chamber of the eyeball, and in the retina. In whooping cough central disturbances of vision have been observed, such as homonymous hemianopsia, probably from cerebral hemorrhage; in some cases an optic neuritis has been present, while in others the children have lost their sight without ophthalmoscopic changes. In addition to the involvement of the optic nerve, secondary to a cerebral condition, paralysis of the III, VII and other cranial nerves have been seen.

MUMPS

Conjunctivitis is not unusual in the course of mumps and is mild in character. Iritis[1] occurs in either an acute, benign form or a chronic and more severe form. Of much greater importance, however, is the inflammation of the optic nerve which has been observed after this infection. This is probably due to a toxin of the bacteria which produce the parotitis, and like involvements of the optic nerve after other infectious diseases, the optic neuritis does not develop until the general disease has been recovered from. It is, in other words, a late complication. The optic neuritis is either a mild retrobulbar neuritis or a severe neuritis which leads to optic atrophy. Paralyses of the ocular muscles have also been observed after mumps. The best-known complication is inflammation of the lacrymal gland. It presents the characteristic swelling in the outer half of the upper lid and both sides are usually affected. The involvement of the lacrymal gland usually follows that of the parotid by a number of days, though sometimes it may be the first sign of beginning mumps. The disease is not severe and does not lead to suppuration.

INFLUENZA

The eye is affected in influenza either through the direct action of the influenza bacillus, as in conjunctivitis, or due to a toxin action as in retrobulbar neuritis and in paralysis of the accommodation. The frequency of ocular complication, according to Groenouw, is about 7 per cent. This author collected the reports of 186 cases and found the cornea and the conjunctiva to be most frequently affected; next in frequency came the uvea and the ocular muscles and then the optic nerve. As regards the lids, lid abscesses seem to be unusually frequent. They occur a number of weeks after recovery from the general infection. The conjunctiva when affected shows hyperemia and lacrymation, rarely a true conjunctivitis. This may be the first symptom of influenza and probably in these cases the conjunctiva has been directly infected by the influenza bacillus.

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[1] Tronyo, Thése de Lyon, 1911.

Influenzal conjunctivitis has the characteristics of a catarrhal conjunctivitis which is limited to the conjunctiva of the lids and to the retrotarsal folds. The influenza bacillus belongs to the group of hemoglobin bacteria of which the Koch-Weeks bacillus is a member. Sometimes the conjunctivitis may be severe and have purulent characteristics. Membranous conjunctivitis has been observed after influenza and in some of these cases the eye has been lost.[1] This bacillus and the closely related pseudo-influenza bacillus have frequently been found in conjunctival discharge. While the infection is probably by direct contact, it is probable that some cases result by extension from the nasal mucosa. Dacryocystitis is usually a secondary infection to that of the nose, and the lacrymal gland has become inflamed in a number of cases. The cornea is not infrequently involved in the form of herpes, usually on the third to the seventh day of the disease; it is usually one-sided and is sometimes accompanied with herpes of the lids. The course is favorable. A similar disease, superficial punctate keratitis; has also been seen. Pflueger reported on 30 cases of parenchymatous keratitis after influenza. The uvea is affected as a mild iritis or a severe metastatic choroiditis. The iritis usually begins not before one month after the onset of the attack. It affects one or both eyes, sometimes with posterior corneal deposits, fibrinous exudation or hypopyon. The course is apt to be very prolonged, though complete recovery frequently ensues. Groenouw states that 20 cases of metastatic ophthalmia after influenza have been reported. Of 11 cases, 9 were limited to one eye, and in 2 both eyes were affected, though both of these patients lived. The bacteriological examination of the eyes which were enucleated showed that the infection was produced by the ordinary pus producing organisms and not by the influenza bacillus.

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[1] Coppez, Rev. d'opht., 1889.
Knapp, Arch. of Ophth., Vol. XXXIII, p. 386, 1904.

Hemorrhages have been observed in the retina and in the vitreous. As to diseases of the optic nerve, Uhthoff found 72 out of 253 cases of optic neuritis due to grippe. Groenouw has studied 67 cases and comes to the following conclusions: The optic nerve is affected usually in the period of convalescence in the second week or later; both eyes are more frequently affected than one. The clinical picture is that of an optic neuritis and the ophthalmoscope shows changes at the disc. A frequent symptom has been a distinct star figure in the retina at the macula lutea. Retrobulbar neuritis with slight or no changes in the eye grounds is unusual. The visual field frequently shows a central scotoma; in other cases concentric contraction. As for the outcome, a part of the eyes obtained fair or good vision, while in some cases very marked loss of vision or even blindness has been observed. The course of the inflammation is a slow one and usually two or three months are consumed. A distinct neuritic optic atrophy remains. Frequent associated symptoms are headache, trigeminal neuralgia, and pain in the orbit on moving the eye. Most cases of optic neuritis after influenza are infectious, produced by toxins, just as the same cause affects other sensory and motor nerves after influenza.

As the nasal sinuses are involved in influenza, it seemed natural to believe that the optic nerve becomes infected secondary to a sinus involvement. The following case which the writer[1] had the opportunity of observing, however, does not bear this out. A patient with a typical optic neuritis and a star figure in the macular region in both eyes in whom the influenza bacillus was isolated from the nose, gave on lumbar puncture the following findings: cell count, 21 lymphocytes; globulin +; liquor was sterile; the Wassermann was negative both for blood and for liquor. As there was some discharge from the posterior ethmoidal cells, these were opened without any effect on the course of the optic neuritis which ran the usual course of these cases, namely, recovery after three to four months. The spinal fluid changes slowly cleared up entirely.

Tenonitis after grippe has been observed in 11 cases.

Paralyses of the ocular muscles are frequently observed after influenza. Groenouw found that the time of onset varies from a number of weeks to months; improvement or recovery takes place. The underlying pathological processes are difficult to explain. It is possible that there are complicating diseases of the brain, polyencephalitis superior hemorrhagica or multiple neuritis. The paralysis may involve any one of the muscles or groups of muscles. The paralysis of the III nerve is usually one-sided or only a few of the muscles which are supplied by this nerve are affected. Paralysis of the accommodation has been frequently observed. It is always bilateral, and the pupils are unaffected. The prognosis is good. Paralysis of the VI nerve is relatively frequent. It is usually one-sided, while the IV nerve is but rarely affected. In addition to these simple paralyses, cases have been observed where a number of nerves have been involved, and typical acute superior polyencephalitis occurred. Mention should be made of the frequency of neuralgias in the course of influenza. The supraorbital branch of the V nerve seems to be frequently involved, possibly through involvement of the nasal sinuses.

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[1] Knapp, Arch. of Ophth., Vol. XLV, p. 247, 1916.

DIPHTHERIA

Diphtheritic diseases of the eye occur from the direct action of diphtheria bacilli with or without associated pyogenic organisms in the conjunctiva, or from the action of toxins which produce a paralysis of the ocular muscles or an optic neuritis. The conjunctiva is usually primarily affected and can secondarily involve the nasopharynx. The reverse is unusual. The clinical appearance of this conjunctivitis is a varying one, as the symptoms are those of a catarrhal or of a membranous conjunctivitis. In other cases which suggest diphtheria diphtheritic bacilli were not found, but ordinary organisms.

H. W. Wooton[1] divides cases of conjunctivitis occurring with diphtheria into two classes: (1) those in which the Klebs-Loeffler bacillus is the organism; and (2) those in which the streptococcus is found. In the first the prognosis is more favorable; recovery with good vision from the conjunctivitis generally takes place. In the pure cases of this type secretion is scanty or absent. Treatment by antitoxin systematically and locally is of great value. In the second variety secretion is more or less abundant, antitoxin is of no value and the prognosis is bad. Membranes are present in both types, not necessarily well-developed. They are never abundant in the streptococcic variety. The latter is probably a mixed infection and in some of the cases both the diphtheria bacillus and the streptococcus are found present.

Primary disease of the cornea does not occur. The lacrymal sac may be involved as a complication from a lesion of the neighboring mucous membrane. Abscesses of the orbit have been observed following diphtheritic inflammation of the conjunctiva.

Optic neuritis is an unusual complication. In Uhthoff's series of 253 cases it was present in only 6. The neuritis occurs only in the severe diphtheritic infections and a number of weeks after the onset of the disease. Just as the various muscles of the body are affected, those of the eye and particularly the ciliary muscles are frequently involved. The frequency of post-diphtheritic paralysis varies from 3.4 to 6.8 per cent. According to Goodale,[1] in 1071 cases of diphtheria, paralysis occurred in 125. Of these, the accommodation was affected in 56 and in 26 the external muscles were involved. The severity of the infection has no reference to the incidence of the paralysis, and it is not infrequent for the oculist to find a paralysis of the accommodation after a comparatively mild angina. On the other hand, paralyses of the external ocular muscles seem to occur in severe cases of diphtheria. The paralysis of the accommodation is usually noted three and one-half to four weeks after the onset of the disease. The patient is usually convalescent, and in the case of children the condition is discovered on their return to school. The paralysis is usually not complete and always affects both eyes. The affection is found nearly exclusively in children under fourteen. The pupils are generally normal, exceptionally they are dilated and the light reaction is sluggish. A frequent complication is paralysis of the soft palate. Paralyses of one of the recti muscles was found in 11 per cent. of the cases of paralysis of accommodation; the prognosis is good and recovery occurs after a number of weeks. The external ocular muscles may be affected in diphtheria without associated paralysis of accommodation. A striking characteristic is their sudden onset and disappearance. A number of muscles may be affected in turn. This paralysis occurs like the paralysis of accommodation in the third to the sixth week after the beginning of diphtheria. The prognosis is good. The most frequently involved muscle is the external rectus of either one or both sides. Complete paralysis of the oculomotor nerve was observed only in one case; as a rule only some of the muscles supplied by this nerve are affected. Ophthalmoplegia externa has been observed a number of times and the ophthalmoplegia has been so complete that the eyes were absolutely immobile. The inner muscles of the eye were not involved. In addition to the ocular paralysis, there was a paralysis of the soft palate and of the facial nerve with ataxia of the extremities. It is of interest to note that the use of antitoxin has not diminished the frequency of these muscular paralyses.

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[1] Personal communication.

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[1] Goodale, Diphtheritic Paralysis, Brain, p. 282, 1896.

ERYSIPELAS

Erysipelas, an infection of the subcutaneous tissue with the streptococcus, is of considerable importance for the eye, as it may begin in the conjunctiva or in the lacrymal passages. As it usually begins in the face, the eyelids become involved and they are then enormously swollen, red and covered with vesicles. After recovery ptosis may remain for some time and repeated attacks of erysipelas can produce a permanent thickening of the lids. Subcutaneous abscesses and gangrene of the lids have been observed and the eyeball itself or the orbit may be secondarily involved. The conjunctiva and eyeball are usually not involved in erysipelas of the lids, although in some cases severe keratitis has been reported. Erysipelas of the lids may lead to an involvement of the lacrymal gland. The abscess of the orbit following erysipelas of the lids is of serious importance to the optic nerve, as the optic nerve or its vessels may be affected either by the inflammation or by compression, and sight is lost either early or after a number of weeks. Ophthalmoscopic examination usually shows optic atrophy with unusually narrow arteries which are outlined with white lines or are completely obliterated. In some cases inflammatory symptoms are present in the fundus or there is the ophthalmoscopic picture of embolism. Thrombosis of the central vein has also been observed. Orbital abscess has led to death in 25 per cent. (Schwendt).[1] Aside from diseases of the orbit with involvement of the optic nerve, the general septic infection may lead to an inflammation of the optic nerve. Cases of retinitis and of inflammation of the uvea have also been reported, and ocular muscle paralyses after erysipelas are infrequent.

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[1] Quoted from Groenouw.

Inflammations of the eyes have sometimes been benefitted by intercurrent attacks of facial erysipelas. Thus in trachoma the granulations and the pannus have been improved and other diseases of the cornea have been aided.

SEPTICO-PYEMIA

Pus-producing organisms are never exclusively pyogenic, but rather phlogogenic, inducing inflammatory processes which vary in the frequency in which they produce pus. The bacteria which are of greatest importance for the eye are the staphylococcus, the streptococcus, the pneumococcus, the gonococcus and the bacterium coli commune. As a result of a local pyogenic infection, bacteria and their poisons may gain access to the blood and lymph channels and so invade the general circulation. If they invade the eye, they give rise to a number of distinct clinical types. The cause for these metastatic pyogenic eye diseases are puerperal, wounds in general, cryptogenetic (so-called when the way of infection is not clear) and certain infectious diseases. The ocular diseases are clinically divided into metastatic ophthalmia and septic retinitis.

Metastatic Ophthalmia.[1]—This condition is the more important of the two as it usually entails serious damage to the eye and when both eyes are affected the patient usually dies. The purulent process is generally not limited to the retina and often the disease appears under the picture of purulent internal ophthalmia or panophthalmitis, where the choroid and often the ciliary body are also involved. Investigations have shown that in most cases the embolic process affects principally the retinal blood vessels and then extends from this layer to the other membranes of the eye. The complicating iridocyclitis may be either independent or an extension of this condition. Metastatic ophthalmia is sometimes the only manifestation of the septic infection of the blood, which may be a very severe one. On the other hand, in some cases we find mild infections which are limited to a certain part of the retina or appear in multiple foci.

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[1] Leber, Netzhaut, Graefe-Saemisch-Hess, 2d ed., Vol. VII.

The first symptom pointing to an involvement of the eye is a loss of eye sight, which may rapidly become total, but this visual disturbance is never so sudden as in cases of closure of the retinal artery. Pain is not a prominent symptom at the beginning, and occasionally the entire disease is unassociated with pain. The early stages are usually not observed with the ophthalmoscope; later the rapidly increasing opacities in the refracting media makes an examination difficult. Where this examination has been possible, extensive opacities and small hemorrhages are found in the retina,the optic nerve is red with blurred margins, and the veins are congested. The exudates in the retina increase, the vitreous opacities become denser, and after one or two days no details in the fundus can be seen, and the vitreous gives a yellow reflex. The cornea generally clouds, hypopyon develops and a purulent iritis is present. The swelling of the lids and of the conjunctiva, with infiltration of the orbital tissues and exophthalmus give all the symptoms of a panophthalmitis. The pus perforates usually in the part of the sclera near the insertion of the recti muscles, and the eye ball then shrinks. There are all gradations in rapidity and severity of the process. In some cases the process may be insidious and two to five weeks may pass before these final symptoms appear. In other cases the external symptoms on the part of the lids and the orbit are absent and the vitreous exudate (so-called vitreous abscess) shrinks so that the eye becomes smaller and the cornea remains comparatively transparent. Exceptionally some sight is retained after a distinct purulent process has developed. The course is usually very acute; in the puerperal form, panophthalmitis is the usual outcome and a shrinkage of the eye without preceding panophthalmitis has not been observed.

In the surgical variety a more protracted course develops and the condition may pass directly into phthisis bulbi. The latter is more frequent (30 per cent. of the cases) when the infection is of internal origin. In these cases the pneumococcus is usually the organism. In the cases of cryptogenetic origin the symptoms are less marked. The exudates become absorbed and there is a contraction of the purulent vitreous infiltration and the eye shrinks.

As regards the anterior part of the eye, the conditions vary, depending on whether an iridocyclitis is present or not. In embolism of the anterior ciliary arteries there are two remarkable conditions, namely, the ring abscess of the cornea and the spontaneous perforation of the sclera. The development of the ring abscess of the cornea has been explained by Fuchs in cases after injuries and operations and in cases of metastatic origin. It results through an enormous development of bacteria in the anterior chamber, probably also in the vitreous and in the lens. Intense toxic action causes a total necrosis of the cornea which, by a demarkating purulent infiltration, becomes sequestered and sloughs. The spontaneous perforation of the sclera takes place regularly at a definite point directly posterior to the insertion of one of the recti muscles. This, in most cases, occurs unusually early and Leber suggests that it is not due simply to a purulent softening of the scleral tissue, but that the purulent process has followed some of the preformed paths in the sclera as the emissaries of the anterior ciliary arteries which pass from the tendons of the recti muscles to the sclera.

Metastatic ophthalmia can be differentiated from cases of orbital abscess and of thrombosis of the ophthalmic veins secondary to a cerebral sinus thrombosis, by the degree in which the eyeball itself is affected.

Prognosis as to life in the bilateral cases is very bad. According to Axenfeld, the mortality in the bilateral cases is 85 per cent., while in the one-sided cases it is 21 per cent. Metastatic ophthalmia occurs in the various forms of pyemia. Groenouw has collected 166 cases in which puerperal pyemia was represented in 76, surgical in 60, cryptogenetic in 30; the form following infectious diseases is not included. Puerperal pyemia is the most frequent cause for metastatic ophthalmia. This is due to the presence of an ulcerating endocarditis, the tendency to the formation of thrombi in parturition and the kind of infectious agent, namely, the streptococcus. The parturient woman is usually in her second or fourth labor. The infection takes place during labor or somewhat later and the eye trouble appears in the first or second week of the disease, though there have been cases known in which it has occurred as late as the seventh. Of 69 cases collected by Leber, 42 were one-sided, and in 27 both eyes were involved. The prognosis for those with a bilateral panophthalmitis as to life was very bad. The parturient patients practically died on an average six days after the involvement of the second eye and after an average duration of the sickness of two weeks. In the one-sided ophthalmias the mortality, according to Groenouw's statistics, is 58 per cent. Endocarditis was absent in all of the cases that recovered, while it was present in more than half of the fatal cases.

Under surgical pyemia is understood the infections which result from injuries and operations and local suppuration, the latter when they are not traumatic but arise from the mucous membranes of the digestive, respiratory or urinary tracts. In this group belong the infections from the female generative organs which are not puerperal in nature. In 53 cases (quoted from Leber) the affection was bilateral in 16, one-sided in 37. Of the former, 75 per cent. died; of the latter, 54 per cent.

Cryptogenetic pyemias are febrile conditions with the presence of purulent metastases in whom the original focus of infection is not determined. They include cases of ulcerating endocarditis, acute articular rheumatism, typhoid fever, meningitis and miliary tuberculosis. A striking characteristic of this group is that nearly half of all those affected are under twenty years of age. The eye trouble begins usually at the end of the first or the beginning of the second week. In 27 cases both eyes were affected in 12, one eye in 15. The mortality in the former was 83 per cent., in the latter 33 per cent. Endocarditis was very frequently present.

Retinitis Septica (Roth).—In septic diseases of various origins multiple retinal hemorrhages and white degenerative areas are found in the retina more frequently than the just described metastatic purulent ophthalmia. These are of small size and probably resemble the multiple small hemorrhages which are observed under these conditions in the skin and in the internal organs. They are not of particular importance for the eye, as the sight is not very much affected, and because of the very severe general sickness of the patient which generally results in early death. At the same time they have great diagnostic and prognostic significance. Litten found in 35 cases of septic disease this condition present in 28, in other words, 80 per cent.

The eye usually presents no external changes, except possibly a few hemorrhages in the conjunctiva and rarely in the iris. The disc and the retinal vessels are normal. The hemorrhages are the principal change. In early cases they appear isolated and occupy the posterior segment of the retina. They are usually round and do not necessarily lie near the blood vessels. The white spots often occupy the center of a round hemorrhage and sometimes appear without the hemorrhages. If the disease lasts for a long time, the hemorrhages and the white spots gradually disappear and new ones appear in other places. There is no transition to metastatic ophthalmia and the two diseases, though their origin is somewhat similar, have a different mode of onset. The choroid is regularly free from any changes.

Leber states that these changes are now regarded as due to soluble bacterial toxins, but this view is unsatisfactory as it does not explain the appearance in discrete areas with apparently normal intervening vessels. General hyperemia is not a characteristic of the findings. An additional feature in the ophthalmoscopic picture is the ganglioform thickening of the nerve fibres. This condition is not thoroughly understood and is probably a retrogressive metamorphosis. The value of this ophthalmoscopic picture in septic diseases is of importance in the diagnosis of typhoid fever, acute miliary tuberculosis and tuberculous meningitis. It occurs in all septic diseases, particularly in puerperal fever. Frequently endocarditis is present. In the above-mentioned diseases these changes have been very rarely observed, particularly in typhoid fever. In the rare cases in which hemorrhages occur in the retina in acute miliary tuberculosis, miliary tubercles in the choroid may be present and in tuberculous meningitis the presence of papillitis is of importance.

TETANUS

As tetanus principally affects the muscles of the body, we sometimes find that the muscles of the eyes are involved. Thus spasms of the orbicularis muscles is sometimes the first sign of beginning tetanus. Spasms of the muscles of the eyeball have been reported by Fromaget. In other reports paralysis of the oculo-motor nerve, ptosis and paralysis of other muscles have been mentioned. Vision is as a rule not involved. The eye may sometimes be the site for the entrance of the tetanus poison, though injuries of the eyeball itself rarely give rise to tetanus and injuries of the surrounding parts of the eye and of the orbit are much more frequent. There are nine cases (Groenouw) of injuries to the eye which have been described. In three cases the injury was a blow with a whip against the eye. In others the injury was produced by a rocket, a shot and a cataract operation. Of these nine cases two recovered after three to four weeks, while all the rest died. Fromaget[1] has given a complete report of the cases of tetanus resulting from injuries to the orbit and the surrounding parts of the eye. The causes in certain cases have been operations on the orbit and injuries.

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[1] Archives d' ophthalmologie, 14, No. 11, p. 657.

SYMMETRICAL DISEASE OF THE LACRYMAL AND SALIVARY GLANDS (Mikulicz Disease)

This is a chronic affection occurring in patients of middle age, in which, without a general disturbance of health, the lacrymal glands and the various salivary glands slowly enlarge. The involvement of these glands is often an irregular one. The swellings are painless and most of the symptoms are due to the mechanical interference which they produce. Jakobaeus[1] finds that a cause is not always the same and distinguishes between a chronic inflammatory group and a leukemic or pseudo-leukemic group. In the former tuberculosis and syphilis are important factors. Prognosis is good unless there is general involvement of the lymphatic glands and changes in the blood. Good results have been obtained in treating the leukemic cases with the X-ray. Other authors believe that this condition has nothing to do with tuberculosis. The pathological examination shows the presence of adenoid tissue which apparently causes an atrophy of the specific gland elements. The course is a favorable one. On account of the mechanical discomfort the lacrymal glands have been extirpated in a number of cases. In some cases arsenic has been of benefit. It is well to consider differentially leukemia and pseudo-leukemia.

MALARIA

The forms of eye lesions which occur in malaria differ with the severity of the malarial infection and consequently with the localities in which the malaria occurs. In this country where malarial fever is frequent, though comparatively mild, the only characteristic eye lesion is keratitis, while in places where the malaria infection is severe, like in the tropics, optic neuritis, retinal and vitreous hemorrhages and effusion into vitreous have been reported. Davis[1] states that ocular complication in malaria occurs when the estivo-autumnal parasite is found and the fever is of the tertian type. The diagnosis of malaria in the absence of typical febrile attacks is difficult and depends on the presence of an enlarged spleen, melanemia and the plasmodium in the blood.

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[1] Jakobaeus, Kl. M. f. A., p. 213, 1910.

An intermittent ophthalmia is described, consisting of attacks of hyperemia of the conjunctiva and of the eye lids which occur during the febrile paroxysm or replace it. This is accompanied with photophobia and tearing, but typical only, according to Groenouw, when neuralgia is present. Between the attacks the eye is frequently normal or the conjunctiva shows slight redness.

The cornea is affected in the form of herpes and of interstitial keratitis. Kipp[2] found malaria present in 90 per cent. of the cases of dendritic keratitis. The keratitis comes on a few days after the paroxysm of malarial fever, usually in only one eye, and the attacks recur in connection with chills and fever. The corneal lesion is nearly always of the type of dendritic keratitis with photophobia, lacrymation and corneal anæsthesia, and sometimes severe supraorbital neuralgia. The amount of congestion varies and according to the degree of infection, corneal ulcers, iritis and hypopyon develop. The duration is usually from two to four weeks, with a disposition to relapse with each subsequent malarial attack. Ellett[3] confirmed the frequency of dendritic keratitis as due to malaria and succeeded in finding the plasmodium in the blood of his patients. Interstitial keratitis has been observed a number of times, principally in the regions where malaria is prevalent.

[1] Davis, Ophth. Year Book, p. 234, 1910.

[2] Kipp, T. Am. O. S., 1899

[3] Ellett, Ophth. Record, 1899.

During the febrile attacks the pupil has been reported dilated, a congestion of the iris or even iritis may be present, with severe pain. A curious whitish infiltration of the vitreous has been observed[1] which affected one eye after the other; it subsequently cleared up completely or partially.

Retinal hemorrhages are not infrequent. They occur during the attacks of fever or in the later stages of anemia and cachexia. The hemorrhages are situated at the posterior pole and both eyes are generally affected. Absorption takes place and no damage results. Vitreous hemorrhages have also been noted. The optic nerve is affected in the form of optic neuritis. Uhthoff found 17 out of 253 cases which were due to this cause. In the tropics Sulzer (*l.c.*) found hyperemia of the nerve-head during the attack of fever in 20 per cent. of the cases. Optic neuritis in malaria is characterized by swelling, a dark reddish-gray color, and by opacities of the surrounding retina with dilatation of the veins. In a third of the cases hemorrhages have been observed in the periphery of the retina. The prognosis is favorable and recovery takes place in most of the cases. It has been suggested that the optic nerve changes in malaria have sometimes really been due to quinine poisoning; a point in differentiation is the great congestion of the disc found in malaria.

Trigeminal neuralgia is frequently due to malaria. The pain occurs usually in daily attacks but without fever; it is sometimes accompanied by swelling of the lids and redness of the conjunctiva.

GONORRHŒA

The gonococcus generally affects the eye in the form of a **conjunctivitis** which is the result of a direct infection whereby the conjunctiva comes in contact with gonorrhœal pus. The organism is a diplococcus, shaped like a coffee bean; it is intracellular, and is Gram-negative. These characteristics are generally sufficient to detect the gonococcus microscopically for practical purposes. The other Gram-negative diplococci which exceptionally may be found present in acute conjunctivitis are the meningococcus intracellularis meningitidis and the micrococcus catarrhalis. They can not be definitely separated except by culture, though there are some slight morphological differences. The meningococcus is of variable size, and is generally larger than the gonococcus, but is not so typically kidney-shaped. The micrococcus also varies in size and is usually not intracellular. Elschnig[1] does not think that the morphological differences—the Gram-negative, intracellular, kidney-shaped characters—are sufficient to identify the gonococcus, and that a culture is absolutely necessary.

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[1] Seely, T. Am. O. S., 1885.
Sulzer, Kl. M. f. A., Vol. XXVIII, 1890.

Notwithstanding the frequency of urethral gonorrhœa, the eye is seldom affected. Gonorrhœal conjunctivitis occurs in the new-born and in adults. The clinical features of these two forms differ in a number of important respects. The adult form is much the severer as the infection is usually derived from a recent and active urethritis and begins in only one eye.

The clinical course is characterized by swelling and infiltration of the conjunctiva with occasional croupous membranes, and a clear watery meat-infusion-like discharge. This stage is followed by the discharge of creamy pus (pyorrhœa). The swelling diminishes and the upper tarsal edge shows papillary hypertrophy. In the second week the discharge gradually diminishes, and the disease assumes a catarrhal character. The important complication is the corneal affection which may be severe or benign. The former occurs in cases with tremendous infiltration of the conjunctiva and the formation of membranous sloughs. The cornea shows uneven stippling and a grayish opacity; this spreads rapidly and the corneal tissue melts away, leaving a total iris prolapse. In the second and more frequent corneal complication a superficial grayish infiltration occurs at a short distance from the corneal margin, which turns yellow and an ulcer develops. Hypopyon may be present. The ulcer does not extend, but penetrates into the depth and may perforate with subsequent iris prolapse, and an adherent leucoma results.

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[1] Elschnig, Gonorrh. Erkr. d. Auges, Handbuch d. Geschlechtskr., Wien, Hölder, 1910.

A mixed infection changes the clinical picture to that of a serpent ulcer, or the infection gains access to the interior and causes purulent iridocyclitis or panophthalmitis. Small abscesses may form at the lid margin and the preauricular gland suppurates. The prognosis depends on the stage that treatment has been begun. Notwithstanding the best of care, the infection is so severe in some of these acute cases, that one or both eyes are lost. The disease is always a very serious one, though there are also milder forms with the clinical symptoms of the ordinary catarrhal conjunctivitis, and where the diagnosis can only be made microscopically.

In the new-born the infection occurs during birth. It is less acute as the gonorrhœal process in the mother is not a recent one, the reaction on the part of the conjunctiva is less (swelling and infiltration) and there is not the same danger of a corneal complication. Both eyes are always involved. The infection occurs not later than the third day. The process is mild; diphtheritic exudates are absent. Severe corneal complications only occur in badly-treated cases or in babies that are in poor health. The corneal complication does not differ from that of adults. A lid complication sometimes appears from ectropium of the swollen conjunctiva which leads to paraphimosis; this can be avoided by care. An important complication is a secondary infection with streptococci, which leads to purulent necrosis of the eyelids. General gonorrhœal infection has been observed.

An interesting complication of gonorrhœal infection in these babies is the occurrence of metastatic joint inflammations. It can be stated that from a prognostic standpoint all cases of gonorrhœal conjunctivitis of the newborn, in which proper treatment is instituted before the cornea becomes involved, get well.

In these new-born babies there are a variety of conjunctival infections which are milder than the above, and in some cases are not produced by gonococci. In these the discharge is not purulent, but more of a mucous character, and the conjunctiva is not as reddened and swollen. Various statistics have shown that about 50 per cent. were caused by non-gonococcic organisms, such as staphylococcus, streptococcus, micrococcus catarrhalis, bacterium coli, diplococcus pneumoniæ. Axenfeld found 273, (59 per cent.) out of 464 gonorrhœal; Elschnig 21 (51 per cent.) of 41; Stadfeldt 182 of 332. Groenouw has shown that from the clinical picture alone we can not say that the gonococcus is present. In fact, quite a number of other organisms can produce a conjunctivitis at this time of life. Most of the cases of this class are characterized by a shorter and milder course and the absence of corneal complications. In the new-born, not only do we find typical gonorrhœal conjunctivitis, but a conjunctivitis of varying degrees of severity. The prognosis is good, especially if the gonococci can not be found. It has been claimed that the use of 2 per cent. silver nitrate solution as a prophylactic is too irritating and produces quite a marked catarrhal inflammation of the conjunctiva. Under these circumstances milder substitutes are at present in frequent use. Probably the most important feature is the cleansing of the eyes with separate materials, and care being taken that none of the water in which the child is bathed gains access to the eyes. In many cases at any rate the prophylactic use of silver drops is not necessary.

In most cases of gonorrhœal conjunctivitis of the newborn, the eyes become affected by the discharge from the genitals of the mother. The discharge comes in contact with the eyelids; then, as the eye lids are opened, the infection gains access to the conjunctival sac. This is the usual cause in the cases which develop from the third to fifth day. If the inflammation begins after the fifth day, the infection must have taken place subsequently to birth. There are a number of cases on record where children have been born with eyes already affected with gonorrhœal conjunctivitis. In these cases the labor was an unusually protracted one and the rupture of the membranes took place very early. The antepartum infection has been studied by Stephenson and Ford[1] who found only 10 cases that stood the test of careful examination; in these the child was born with an active gonorrhœa, and the water had escaped at a varying time before. Infection through the intact membranes is not demonstrated. Cases of late infection, those that occur after the fifth day, can not be regarded as gonorrhœal, as the bacteriological proof is not given.

There is a purulent conjunctivitis in young girls between two and ten years of age, who are suffering from a vaginal discharge. As a rule this is a true gonorrhœal infection, though there is vaginal discharge in children which does not contain gonococci and may produce a mild inflammation of the eyes. It is not infrequent that a discharge containing the gonococci is transmitted from an infected eye to another; it is in this way that physicians and nurses become infected. In the examination of babies with conjunctivitis care must be taken that the sudden opening of the lids is not followed by a spurt of secretion which might endanger the physician's eyes.

Sequelæ.—Elschnig mentions cases of lid gangrene in infants which probably were due to mixed infections. Infants with gonorrhœal conjunctivitis often have rhinitis; it is difficult to demonstrate gonococci in the nasal discharge. A gonorrhœal infection of the mouth has been observed (stomatitis). Inflammation of the joints has often occurred, though generally only in infants. This usually comes on in the second or third to fifth week, and the joints swell, become red and painful. The joints affected are the knee, hand or foot. Recovery usually results. In the joint-fluid gonorrhœal organisms have been found. The gonococci must penetrate the conjunctiva and reach the lymph and blood vessels in these cases. General septicæmia has been reported after gonorrhœal conjunctivitis.[1] In addition to joint lesions there were cutaneous eruption, septic endocarditis and pericarditis.

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[1] Stephenson and Ford, The Ophthalmoscope, April, 1906.

Metastatic Gonorrhœal Conjunctivitis.—This is a bilateral catarrhal conjunctivitis which, according to Groenouw, is principally localized in the retro-tarsal folds. It differs from the usual gonorrhœal conjunctivitis through its mild course, inasmuch as it heals in a few days. Furthermore, the discharge is mucopurulent, not truly purulent, and it does not contain gonococci. Joint affections either precede or occur later. Iritis is sometimes present, which is not due to the conjunctivitis, but appears as an associated metastatic condition. With the onset of a new urethral infection, there may be a relapse of this form of conjunctivitis. There are very few bacteriological findings in endogenous conjunctivitis reported in literature. According to Morax and Axenfeld, this is analogous to the negative findings in gonorrhœal arthritis. These authors believe that the gonococci may irritate the mucous membrane by their presence in the conjunctival tissue without actually being present on the surface. In the histological examination of the conjunctiva in endogenous gonorrhœal conjunctivitis a number of gonococci have been found in the conjunctival tissue. It seems therefore that they may be present deep in the tissues and not appear in the conjunctival smear. Heerfordt[2] found 23 cases of metastatic conjunctivitis among 2300 cases of gonorrhœal urethritis. These occurred principally in the bulbar conjunctiva, developing under the conjunctival surface, often with the formation of phlyctenules, so that he gave this affection the name of "sub-conjunctivitis epibulbaris gonorrhoica." Pincus[1] has described two cases in which **keratitis** occurred apparently in the form of a metastatic inflammation of gonorrhœal origin. The clinical characteristic was a strictly epithelial inflammation in the form of epithelial defects whose origin seemed to be vesicular. A striking feature was the tendency of this epithelial detachment to spread, though the healing was as rapid as in epithelial erosions. In some cases there were also opacities in the deeper layers of the cornea. In order to make the diagnosis of a metastatic process, the following conditions must be fulfilled. Gonococci must be present in the discharge of the urethra or vagina; there must be other manifestations of a systemic gonorrhœal infection, such as an inflammation of the joints, tendon sheaths, muscles, endocardium; if conjunctivitis is present an examination of the conjunctival secretion must be negative for gonococci. In Heerfordt's cases the epithelial elevation (vesicle) was a distinguishing characteristic, and this author speaks of a vesicular and parenchymo-vesicular and parenchymatous form and assumes that the keratitis is always secondary to a gonorrhœal sub-conjunctivitis. Pinkus compares the keratitis due to an endogenous gonorrhœal cause with herpetic keratitis; he was never able to find any secondary disturbances present in his cases, though the extent of the epithelial detachment is much greater and the recovery is more rapid.

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[1] Stephens, Ophth. Record, 1905.

[2] Heerfordt, "Sub-conjunctivitis Epibulbaris Gonorrhoica, Graefe's Archiv., Vol. LXXII, p. 344.

Gonorrhœal Iritis.—Though the proof that a conjunctivitis is a metastasis of a gonorrhœal infection may be difficult to give, there is no question but that a metastatic gonorrhœal iritis is not an infrequent condition. Particularly must this etiology be thought of when the iritis is a frequently recurring one. In the same individual with relapses of a gonorrhœal infection it is not infrequent for the iritis to promptly recur. This iritis is generally found in males. There are usually numerous posterior adhesions of the iris in both eyes; the patients suffer from recurrent articular inflammations. In the acute stage a gelatinous exudate will often be found in the anterior chamber, which practically fills this cavity and in a few days contracts, giving a very characteristic clinical picture. Iritis which is due to this cause is always a serious one, and in the writer's experience it is frequently associated with inflammation of the deeper structures of the eye (vitreous opacities and neuroretinitis). In fact, in cases which apparently respond to treatment and where the iritis disappears promptly, the congestion of the retina will remain for many weeks.

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[1] Pincus, A Contribution to the Study of Endogenous Corneal Infections, Archives of Ophthal., Vol. XLVIII, p. 136.

Cases of iritis due to gonorrhœa usually give a history of one or more attacks of urethritis. Manifestations are present of lesions in the genito-urinary tract which indicate a general gonococcal sepsis. The metastases usually take their origin in abscesses situated in the uro-genital apparatus or from joint affections. Sidler-Hugenin[1] was able to demonstrate gonococci in the blood of 5 of his 12 cases, while the gonococci were cultivated from the contents of the anterior chamber in only 1 case out of 6. This was an unusually severe inflammation. In order to obtain positive findings, this author recommends that the blood or secretion from the anterior chamber be taken during the time of highest temperature. It is furthermore necessary that a large quantity of material should be obtained and, to insure results, the material must be transferred to an incubator as rapidly as possible and the culture media should be warmed before using. In other words, the gonococcus is an extremely sensitive organism, and the technique must be free from faults. Goulden[2] has divided metastatic gonorrhœal iritis into the following varieties: (1) slight iritis, the symptoms are mild with but little tendency to the formation of adhesions and deposits are rarely seen; (2) plastic iridocyclitis which is very much more severe and the more frequent. The symptoms are all marked and there is a tendency to exudation into the pupillary area with the formation of broad adhesions; (3) exudative iridocyclitis. This is a type described by Mackenzie as typical of gonorrhœal iritis, and is characterized by a profuse exudation of lymph into the anterior chamber. This form recovers completely under vigorous treatment. (4) Hemorrhagic iritis, in which form hemorrhages occur into the anterior chamber. These cases with relapsing iritis when reinfected, are probably all the time reinfecting themselves from uncured conditions of the deep urethra and prostate. It has been found that gonococci were present in centrifugalized urine thirty years after the primary infection.

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[1] Sidler-Hugenin, Arch. of Ophth., Vol. XLI, p. 360.

[2] Goulden, R. L. O. H., Vol. XIX, p. 360, 1914.

Acute Dacryoadenitis.—Twelve cases are reported in the literature (Elschnig). In some the adenitis was preceded by a metastatic conjunctivitis with gonococci and in others it appeared independently. In the former the metastatic nature is not proven, as it is much more probably a direct extension, though this is very unusual. In the second group there were no gonococci in the conjunctiva. The bilateral dacryoadenitis in gonorrhœa is characterized by an unusually mild and benign course, recovery taking place in eight to fourteen days.

SPOROTRICHOSIS

Sporotrichosis in the eye is usually due to the sporotrichon Beuermanni. It is often the first localization of the infection. The conjunctivitis shows follicular and ulcerating changes. At the lid margins swellings and ulcerations have been observed and dacryocystitis. Wilder and McCullough[1] reported upon a student who was infected from laboratory cultures of the sporotrichon. The lids became swollen and the palpebral conjunctiva was so swollen that the fornix rolled out in a mass when the lids were everted; grayish-yellow ulcers formed on the palpebral conjunctiva and in the fornix. There were also enlarged follicles and the preauricular glands were swollen and tender. This article reports upon 15 cases found in the literature. Gifford[1] described the ocular lesions in sporotrichosis and Bedell[2] reported a case of conjunctivitis with an area of granulation tissue and numerous brownish concretions in the canaliculi and lacrymal sac which proved to contain the sporotrichon. The characteristics of this infection seemed to be a mucopurulent discharge, a swelling of the conjunctiva, the presence of yellowish follicles and superficial ulcers, together with the involvement of the neighboring lymph glands.

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[1] Wilder and McCullough, J. A. M. A., 1914.

The intraocular forms are not so frequent but are very important. They occur in the course of severe sporotrichosis as iritis, iridocyclitis, keratitis, tumors of the choroid or retina.

The diagnosis can be easily made from the biological examination.[3] The various forms can be produced experimentally in animals; if injected into the blood, interstitial keratitis, iritis and retinitis have been observed. Aurand[4] has inoculated the conjunctiva of the rabbit and has found characteristic changes. Circumscribed tumors appear beneath the conjunctiva without inflammatory symptoms. Inoculation of the cornea produces a keratitis like the aspergillus keratitis or a slowly developing interstitial form. Inoculation into the anterior chamber causes a swelling in the iris which resembles a tubercle or iridocyclitis. Inoculation of the vitreous causes changes in the choroid. The lesions are usually benign and are absorbed. Notwithstanding the specific treatment with iodine, ocular sporotrichosis heals slowly and the intraocular forms are serious.

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[1] Gifford, Ophth. Record, 1910.

[2] Bedell, J. Am. Oph. Soc., 1914.

[3] The conjunctival secretion is spread on ascitic agar or Sabourand's "gélose maltosée" (Morax, Précis d' ophth., p. 184). White colonies develop at room temperature in five to eight days, which soon turn brown or black. Under the microscope the mycelium and numerous spores are visible.

[4] Aurand, Rev. gen. d'opht., p. 246, 1909.

STREPTOTHRICOSIS

It has been known for some time that the lacrymal passages can become stopped up by fungi and concretions in the lacrymal canals have frequently been described. The organisms[1] found in these concretions have been regarded as leptothrix, streptothrix or actinomyces, but cultivation has generally shown the presence of the streptothrix. The clinical picture of the occlusion of the lacrymal passages begins with tearing, the caruncle and the surrounding parts become red. Along the course of the canaliculus an ovoid swelling develops, which can not be reduced though sometimes a little cloudy fluid can be expressed out of the tear points. On incising this tumor, its connection with the lacrymal passages will be demonstrated. On splitting the tear canals yellowish or greenish concrements will be found present. They contain the fungus and calcareous particles and are sometimes surrounded by granulation tissue. The condition is unusual; Groenouw states that 39 cases have been collected from the literature in which actinomycosis was found present in 11. The lacrymal sac is usually not involved. It is striking that even if this condition exists for a long time no suppuration develops, thereby differing from the usual course of actinomycosis. Actinomycosis of the conjunctiva has been described. In one case yellowish granulations were found in the fornix. Concretions in the conjunctiva are sometimes conglomerates of the streptothrix.

Landrio[2] gives a review of the entire subject and finds that the literature contains reports of 19 cases of aspergillus keratomycosis, 20 of sporotrichosis, 1 of keratitis from verticillium, 2 of conjunctivitis from iodium albicans; some more or less severe cases of blastomycosis of the lids, conjunctiva and cornea, several of favus actinomycosis, 23 of concretions in the lacrymal canuliculi observed in France, and 1 case of streptothrix.

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[1] Fungus stains best with Gram's method, and for cultivation it should be grown anaerobically.

[2] Landrio, K. M. f. A., July, 1913.

The eye is sometimes secondarily involved in actinomycosis of the jaws where the eyelids were affected and in one case a retrobulbar abscess developed.

ASPERGILLUS

Infection of the cornea with aspergillus is a well-defined condition called keratomycosis. It is due to the aspergillus fumigatus which is inoculated into the cornea at the time of an injury. The clinical appearance is that of a prominent infiltrate or of a fascicular keratitis. The affected region is peculiarly dry and is outlined by a ring of demarcation; it is cast off like a sequestrum in which the mycelia will be present. Aspergillus infection of the vitreous has been described.

LEPROSY

Leprosy is a chronic infectious disease caused by the lepra bacillus which attacks particularly the skin, the nerves and internal organs. It generally appears in two clinical forms which are often combined—the anæsthetic and the tubercular types.

The anæsthetic type progresses very slowly and after obscure nervous symptoms areas of erythema appear in the skin with hyperæsthesia which gradually are transformed into yellowish anæsthetic areas. In addition to the areas of erythema, there are hard brownish-red infiltrations. The symptoms of the true neuritis due to leprosy are very much more marked and consist in neuralgia, hyperæsthesia, trophic disturbances and anæsthesias. In this form injuries occur and the muscles atrophy, which, as far as the eye is concerned, shows itself as lagophthalmus.

The tubercular type presents small hard nodules in the skin of a brownish color which sometimes unite to form diffuse infiltrations. The face and the extensor surface of the extremities are especially liable to involvement. When they appear in the face the hypertrophies in the eye brows, the nose and the auricles give the individual a characteristic appearance. These nodules have also been observed on the cornea. The tumors appear in the internal organs and in the peripheric nerves with resulting change in sensibility. The nodules are either absorbed, disintegrate and form ulcers. The course is a very chronic one.

The eye and its surrounding parts are frequently involved in leprosy. As a rule the condition is localized to the anterior segment of the eye. de Silver[1] found ophthalmic changes in leprosy present in 20 per cent. of the patients. The usual form was an infiltration of the eye brows and lids, though the most important was a tubercular episcleral growth which gradually involved the cornea. The anæsthesia of the region of the brow, conjunctiva or cornea is sometimes present. The fundus is usually not involved and cases of cataract were not more frequent than usual. According to Lyder Borthen,[2] ocular complications in leprosy are a frequent occurrence between two-thirds and three-fourths of the patients being affected. In nearly all of the cases the ocular condition is a bilateral one and in a definite proportion, varying from 30 to 40 per cent., this condition leads to blindness. In the anæsthetic variety more than half of the patients suffer from a paralysis of the frontal muscle and of the corrugator of the eyelids. The face is thereby given a curious immobile expression. The tuberculous type occurs in the forehead in the form of diffuse infiltrations or circumscribed nodules, which is also accompanied by a paralysis of the frontalis muscle. The eye brows are similarly affected and frequently form the very beginning of the condition. The tubercular leprosy begins in the lid as a diffuse infiltration presenting the clinical picture of œdema. The color is a dark red or blue. In addition to the general swelling, nodules can be palpated in the tissues. The margin of the lid is frequently affected and a nodular infiltration or a group of nodules show a tendency to extend along this margin. Michel speaks of the frequency in which these nodules appear at symmetrical parts of the eyelids and that these are often the only manifestations of a facial leprosy. In other cases the lids are affected as an extension from disease of the skin of the face, particularly of the eye brows.

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[1] de Silver, Ophthalmoscope, February, 1908.

[2] Lyder Borthen, Lepra des Auges, Leipzig, 1899, Christiania, 1902.

The hair of the eye brows is lost and the cilia disappear. The eyelids are frequently involved and after the infiltrations and nodules have partly broken down and have formed scars, a deformity of the eye lids results.

Paralytic lagophthalmus is a characteristic picture for the anæsthetic type. This is due to an atrophy of the orbicularis muscle and of the lids.

In the anæsthetic form of leprosy the lids show a reddish infiltration with secondary atrophy, loss of the eye lashes and symptoms of anæsthesia. The facial nerve is generally paralyzed and the orbicular muscle atrophies. The lower lid becomes ectropionated and a keratitis from exposure frequently results.

In both varieties a mild conjunctivitis is present. Occasionally yellowish-brown areas have been observed on the conjunctiva. The leprous tumors are not frequent and are usually a secondary extension from new formations in the neighborhood. They occur in the form of flat infiltrations or nodules of whitish color, which disappear with scarring or form ulcers.

The episcleral tissue is frequently involved; according to Borthen in 41 per cent. in the nodular form of leprosy. In the anterior part of the sclera diffuse infiltrations or small nodules appear which show the characteristics of a phlyctenule or a patch of episcleritis, occasionally with a brownish-yellow discoloration of the conjunctiva. They do not usually form ulcers, and leave a grayish discoloration. The disease is characterized by frequent relapses and by a tendency to extend to the iris and adjoining parts of the cornea.

The cornea is most frequently involved in the anæsthetic type in the form of a keratitis from exposure. The epithelium near the limbus first becomes changed and then a pannus develops in the lower half of the cornea, finally the entire cornea may be involved. According to Borthen, the nodular form of leprosy involves the cornea in 73 per cent., usually complicated with episcleritis or iritis. There are two forms of corneal involvement, namely, the leprous keratitis and the leprous nodules. The leprous keratitis occurs either in the form of a punctate or parenchymatous keratitis, or an opacity in the periphery of the cornea like the arcus senilis. The parenchymatous keratitis is but a further development of the punctate keratitis and is an infiltration of the deeper and the middle layers of the cornea with the leprous type of infiltration. Leprous nodules in the cornea have been repeatedly observed. They are always secondary to lesions at the margin. The process begins with an opacity in the cornea and then a nodule appears of varying prominence, yellowish-gray or red in color, with a gradual diminution toward the sclera and an abrupt limitation toward the corneal center. The entire cornea can be involved, while in some cases the periphery is affected and the center remains free. The condition is not accompanied with pain and always presents complications in the form of iridocyclitis. The nodules may break down and form ulcers or may be absorbed. The prognosis is serious because the deeper parts of the eye are always involved.

The uvea is affected in the tubercular variety more frequently than in the anæsthetic type. Isolated iritis is uncommon and it is usually accompanied with scleritis and involvement of the ciliary body. The condition is always serious, as the pupil becomes occluded and the eye ball shrinks or secondary glaucoma develops. Leprous nodules have been reported in the iris in the form of small gray nodules which appear at the pupillary margin, and larger tumors in the angle of the anterior chamber. Intraocular changes can not be determined, owing to the changes in the anterior half of the eyeball, though they have been described.

In *hookworm disease*, according to Jervey,[1] there are no characteristic eye lesions and those that are present are due to the secondary anemia. Calhoun believes that the serious eye changes are due to a toxin rather than to the secondary anemia.

Anthrax is the result of contact of infected hands with superficially injured places in the skin, and thereby the eye lids become involved either in the form of a pustule or as œdema. The pustule begins as a small red spot which forms a papule and a bluish-red vesicle with blood-stained fluid. When this vesicle ruptures a black, depressed scab develops. The scab is surrounded by an infiltrated area upon which a ring of serous vesicles appear. The surrounding skin is œdematous and the regionary lymph glands are enlarged. The œdema shows a doughy soft transparent œdematous swelling without any symptoms. It becomes red, then vesicles appear which rupture and form scabs. Those who deal with animals are particularly likely to be affected, and the hides of diseased animals have transmitted the contagion. Morax found that in 50 cases of lid anthrax death occurred in 15.

In *bubonic plague* hyperemia of the conjunctiva is one of the most important symptoms.[2] Plague conjunctivitis is characterized by a scanty, thin discharge which is stained brown from extravasation of blood and contains enormous quantities of the plague bacillus. The neighboring carotid and submaxillary glands are often involved. Eye complications occur in 4.3 per cent.; these are usually present in both eyes and consist in panophthalmitis, keratitis and iritis.

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[1] Jervey, J. A. M. A., Vol. LXIII.

[2] Mizuo, Arch. f. Augenh'lk., Vol. LXV.

Trichinosis.—As trichinæ become lodged in the voluntary muscles, they have been found in the ocular muscles. Severe pain is present, particularly on moving the eyes, so that the eyeballs are often rigid. A characteristic of trichinosis is the presence of œdema and is particularly noticeable in the eyelids and in the face. It is apt to be transient and recurs. The œdema may also affect the ocular conjunctiva and the fornix; if the orbit is invaded exophthalmus is present. The intraocular muscles have also been affected, causing a mydriasis with or without accommodative paralysis. This is regarded as due to the action of a toxin (Groenouw). The diagnosis depends on the history of the ingestion of contaminated pork and the demonstration of the parasite in the muscles.

SYPHILIS[1]

Syphilis causes about 2 per cent. of all eye diseases. The eye may be the site of entry for the general infection of the body with syphilis, though much more frequently it becomes affected as a manifestation of the general infection. According to Groenouw, the uvea is the part of the eye most frequently affected, namely in 43.2 per cent.; next come in frequency the optic nerve, 24 per cent.; the ocular muscles 15.5 per cent.; the retina 6.3 per cent.; the cornea 4.1 per cent.; the lacrymal apparatus 1.3 per cent.; the lids and conjunctiva 1.1 per cent.; the orbit 0.2 per cent.; and the sclera 0.1 per cent. Both eyes are affected in nearly one-half of the cases.

[1] Groenouw, Graefe-Saemisch, II ed., 1904.
Dimmer, Syphilis d.Auges, Handbuch d. Geschlechtskr. Hölder, Wien, 1910.
Alexander, Syphilis u. Auge, Bergmann, Wiesbaden, 1889.
Terrien, Syphilis de l'oeil, Steinheil, Paris, 1905.
Leber, Graefe-Saemisch-Hess, II ed., 1916.

Diagnosis.—In addition to the clinical appearance of the ocular lesion, which in many cases is suggestive, and to the general symptoms, the discovery of the spirochetæ and particularly the Wassermann test have been of the greatest aid in diagnosis in ophthalmology as in other branches of medicine. The luetin test, as it is a spirochetal reaction, is even more sensitive than the Wassermann. If the primary lesion is situated in the lids or the conjunctiva, the presence of the spirochetæ can be established with the dark field microscope. The value of the Wassermann test varies somewhat with the frequency in which it is positive. Thus, in inherited syphilis the Wassermann is practically always positive, and it remains so notwithstanding treatment. It acquired lesions the percentage is very much less. It must be always kept in mind that a positive reaction is proof of a syphilitic infection; a negative result is not proof of the converse. Manson, Mackie and Smith[1] state that in the tertiary and late stages of syphilis only 75 and 50 per cent. respectively give a positive result. In interstitial keratitis these authors found the reaction was positive in 88.8 per cent. In iridocyclitis the proportion of positive results varied from 50 to 60 per cent. In choroiditis, of 26 cases 20 were negative. In sympathetic ophthalmia the blood is negative, also in retinitis pigmentose. Inflammation of the optic nerve and retina, 5 positive out of 14. In optic atrophy, all cases of primary optic atrophy were positive. Of the 13 cases of ocular muscle paralysis, 7 were positive, of which 4 were involvements of the III nerve. The 6 negative cases were all paralysis of the VI nerve. Of 6 cases of myopia, with choroiditis, 2 gave a positive reaction. The quantitative Wassermann reaction (Lesser[2]) informs us not only on the exact strength of the reaction and on the intensity of the infection, but on the effect of treatment. Salvarsan has now been on trial sufficiently long to bring out some definite facts. Different from the aryolarsenates, it does not exert any poisonous action on the optic nerve. Affections of the optic nerve and of the nerves to the ocular muscles, the so-called neuro-recidives, are an important feature in salvarsan therapy. These lesions must be regarded as a novelty in the early course of syphilis, and are now regarded as syphilitic manifestations and as evidences of a Herxheimer reaction. They are cured by mercurial treatment and can be avoided by the cautious initial administration of salvarsan.

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[1] Manson, Mackie, and Smith, B. M. J., Feb. 20, 1915.

[2] Lesser, M. m. W., Jan. 13, 1914.

EYELIDS.—*Primary Lesion.*—Among extragenital chancres this location is represented by 3 to 4½ per cent. The primary lesion in the eyelids is situated either in the skin or the conjunctiva, or at the junction of these two structures, the last position being the more frequent, especially near the internal caruncle. It appears as a bluish-red, or a brownish-red swelling. The lid is œdematous, infiltrated, and through the skin the typical induration can be palpated. The lesion ulcerates so that the lid becomes ectropionated and the cilia are lost. The neighboring glands are swollen. The ulcer may extend to the neighboring conjunctiva causing considerable discharge, and the general appearance may suggest a gonorrhœal ophthalmia. There are cases on record of double lid chancres. The course is that the swelling gradually diminishes, and complete recovery takes place. Occasionally the cilia remain deficient, and deformity of the lid results. In diagnosis, a hordeolum or chalazion may be suspected, though the presence of an ulcer with the swelling of the lymph glands suggest the correct diagnosis. A tuberculous ulcer or an epithelioma develops much more slowly, and the age of the patient is also different. A primary lesion in the region of the lacrymal sac may simulate a dacryocystitis.

Cutaneous eruptions on the eyelids in the secondary stage are of slight importance.

There is a form of syphilitic ulcer of the eyelids (rupia syphilitica) which occur in the tertiary stage and is usually an extension from the adjoining skin; it is characterized by the rapid breaking down of the infiltrated tissue.

Tarsitis syphilitica is a slow and painless swelling of the tarsus which involves a part or the entire tarsus. It has been observed in 21 cases (Groenouw 1904). The enlarged tarsus can be palpated through the skin which is red, the conjunctiva is hyperemic and the cilia are defective. The adjoining lymph glands are enlarged. The swelling disappears on appropriate treatment, and may leave an atrophic deformity of the tarsus. The condition occurs sometimes in the secondary stage, though in others the infection is of many years' standing. The age of the patient is usually over thirty and the lues is generally old. There are other specific symptoms, especially gummata of the forehead. Generally, only one lid is involved. If the condition develops acutely, a tumor forms with severe pain and fever. This breaks open and leaves a deep ulcer which may perforate the eyelid. In the chronic form, the swelling develops more slowly, without pain and the skin may be thickened over it.

As a **gumma,** a disease of the eyelids is described which may involve the tarsus and conjunctiva, and lead to extensive destruction of the eye lids throughout their entire thickness. The onset is acute, or there may be a more chronic swelling which suggests a stye or a chalazion. The skin ulcerates and the margins of the ulcer are infiltrated. The diagnosis can usually be made from the history, and the simultaneous appearance of tertiary symptoms in other parts of the body.

CONJUNCTIVA.—*Primary Lesion.*—Primary lesions of the conjunctiva are very unusual. Up to the present date 47 cases (Saemisch) have been reported in which the lesion was situated in the lower retro-tarsal fold or on the conjunctiva of the lower lid in 20 cases; in the conjunctiva of the upper lid or in the upper fornix in 8; in the ocular conjunctiva in 19. The symptoms of the localization to the lower lid are those of gradually increasing irritation, coming on usually in the third or fourth week after infection, with congestion of the conjunctiva and a swelling of the lid. The area affected then develops the characteristic hardness in which is associated the increased size of the tarsus which can be easily palpated through the unchanged skin.

The area of infection presents a dirty white color and is covered with a gray fibrinous membrane. The immediate surroundings present the characteristic hardness. The preauricular and submaxillary glands are enlarged. Other changes in the body are at this point not seen. After a short time other glands enlarge and the cutaneous eruption appears. The lesion usually produces no pain and only slight irritation.

Chancres of the bulbar conjunctiva are infrequent, though they have been described in a number of cases. They are usually situated to the inner and lower side of the cornea. The symptoms consist in photophobia, pain, a definite infiltration of the conjunctiva which increases in size, and is characterized by unusual hardness. A superficial ulceration then develops. The neighboring lymph glands enlarge after fifteen to twenty days; the ulcer heals, the hardened yellowish-red tissue underneath it increases in size, and there is serous infiltration of the surrounding tissue. The process then slowly diminishes, though the site is noticeable by a discoloration for a long time.

Sourdille has reported 13 of these cases, and emphasizes the absence of pronounced ulceration with but little irritation and secretion, but with enlargement of the neighboring glands.

The diagnosis of a chancre of the conjunctiva owing to its infrequency is often difficult. If it is situated on the conjunctiva, the condition may be mistaken for pustulous conjunctivitis, tuberculosis or an epithelioma.

The characteristic induration which can be palpated through the unaffected skin, and the involvement of the neighboring lymph gland, are important points in the differential diagnosis. The prognosis is usually a favorable one, as early treatment causes the lesion to heal, and frequently prevents the generalization of the disease.

Secondary and Tertiary Syphilis of the Conjunctiva.—Some authors have observed an obstinate and tedious conjunctivitis in those affected with syphilis. Michel has described the development of lymph follicles in the lower retrotarsal folds in the early periods of syphilis. This is supposed to differ from the ordinary trachoma by a peculiar anemic appearance of the conjunctiva, and the condition does not improve with local treatment.

The conjunctiva just as other mucous membranes is the seat of **papules** and **gummata** in the various stages of syphilis. Associated with these conjunctival lesions, changes in the other parts of the body are present. These conjunctival lesions are often accompanied by a catarrhal conjunctivitis.

The syphilitic conjunctival **papule** is usually round, varying in size from the head of a pin to a pea. In some cases it is a prominent spot with a curious copper-like discoloration. It is particularly apt to be localized at the semilunar fold and at the caruncle. Wilbrand and Staelin have found papules of the conjunctiva present in 10 per cent. of cases in the early period of syphilis. They usually disappear with general treatment. They resemble phlyctenules, though the age of the patient and the other signs of syphilis confirm the diagnosis. The surrounding tissues are congested.

The development of a **gumma** of the **conjunctiva** is sometimes associated with severe pain and symptoms of irritation. Not infrequently the affected eye presents other syphilitic manifestations in the form of iritis, and in other parts of the body gummata are found in the mucous membranes, in the skin, and particularly in the skin of the lids. The gumma is a sharply defined round swelling, hard, dark red in color and is surrounded by an injected zone. The rest of the eye is not changed, though iritis is not infrequent.

If the tumor is situated in the margin of the cornea, opacities of the cornea are present. Its site is more frequently on the bulbar conjunctiva than on the palpebral conjunctiva. It is usually solitary and most frequently in the region of the insertion of the external rectus. The course is very chronic, and the subjective symptoms are usually absent. For the diagnosis, it is important to remember that it rests more upon other evidences of tertiary lues, its subsequent course and success of treatment, than upon the appearance of the tumor itself. It may at first resemble an episcleritis or a chalazion or epithelioma.

After a primary lesion of the conjunctiva, the affected eye seems to be more susceptible to other syphilitic manifestations as instances of other syphilitic diseases have been found in the same eye, though at a later period, so that a direct extension of the infection has been accepted.

Treacher Collins[1] has reported on a number of cases where interstitial keratitis developed in the same eye after an interval varying from five months to ten years, where previously a chancre had been located in the conjunctiva.

Cornea.—The cornea is involved in the form of a deep-seated inflammation called **interstitial keratitis.** It is a hereditary lesion in the majority of the cases, while it is very unusual in acquired syphilis (3 per cent.). Parinaud believes that this keratitis represents attenuated syphilis of the parents, inasmuch as a certain number of the affected children show no other sign of infantile syphilis and in whom the keratitis is the first manifestation of syphilis. The infantile syphilis of the early months, a period in which the interstitial keratitis is very unusual, corresponds to a period where the disease of the parents is still contagious, while the interstitial keratitis belongs to a later and latent period.

Interstitial keratitis begins with very slight or very marked symptoms of irritation (ciliary congestion, photobia, lacrymation). The opacity of the cornea consists of grayish spots or larger opacities, or of a number of grayish lines which start at the periphery or in the center of the cornea and enlarge until the entire cornea is covered and resembles ground glass. The surface of the cornea becomes dull and stippled. The blood vessels are formed in the deeper layers of the cornea. The limbus become thickened and small red vessels appear to enter the deeper layers of the cornea; they are crowded together and give the cornea a diffuse pinky color (salmon patch). The degree of opacity and vascularization varies in extent according to the severity of the process, and the vascularization may not take place. In some the process is so severe that the cornea become thinned and ectatic. The iris is always involved, and it is often difficult to get the pupil to dilate. No particular changes can be observed in the iris stroma. That the process also involves the ciliary body is shown by the posterior corneal deposits, and the tendency to glaucoma. This latter condition may help in producing the keratoglobus which is seen in the severe cases. The clearing up of the cornea takes place usually slowly from the periphery leaving an opacity in the center of varying degree. When an ophthalmoscopic examination can be made peripheric choroidal changes are seen suggestive of inherited syphilis. The cornea never ulcerates, and its clearing up power is marvellous, though the deep blood vessels can always be made out under proper magnification (Hirschberg). The infiltration from the beginning may not be diffuse, but consists of a number of separate infiltrates with small yellowish spots; this is a very obstinate form. Vossius has described a form where the condition assumes a ring-like infiltration, "keratitis annularis;" the center later on becomes cloudy and the ring disappears. Interstitial keratitis occurs between the fourth and twentieth, usually the tenth and fifteenth year. Sometimes it appears later, between twenty-five to thirty years. In the great majority of cases the cause is inherited syphilis, which serological examinations in recent years have confirmed. The anamnesis (history of abortions in the mother, etc.) and other signs of inherited syphilis are usually present.

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[1] R. L. O. H., 1904.

Collins[1] investigated the children of 12 mothers with inherited syphilis. The children seemed to be free from taint, but an unusually high infant mortality (20 per cent.) suggested some dystrophic influence.

In the **acquired** form the keratitis must not be confounded with that form which is secondary to disease of the uvea or of the sclera. The clinical picture is not very different from the inherited type, except that it is more frequently one-sided. The form of corneal opacity is not characteristic and the development of the blood vessels is frequently defective. The inflammatory symptoms of the conjunctiva are slight and the subjective symptoms are not marked. Corneal ulcers do not belong to this picture. Iritis is not frequently present. Muscular paralysis in the eye of the same side, or in the other eye, have been observed. The course is favorable, though it takes about three months, which is shorter than in the inherited type. The age of the patient is usually between twenty and forty and the infection has generally been at least six months old. Dimmer believes that there is no special form of keratitis following acquired lues, though in general the course is more rapid and the prognosis is more favorable than in the inherited. It occurs either in the secondary or in the tertiary stage. Frequently, there are associated secondary symptoms. Recently the question has arisen whether an injury in a luetic can produce an interstitial keratitis in the injured eye, and later a similar condition in the other eye. This question is still undecided. The keratitis which occurs secondarily to irido-cyclitis is a complication of the latter disease and will be described under another heading. Fisher[2] finds that acquired interstitial keratitis is a very late secondary or tertiary infection affecting usually only one eye. The involvement of the cornea is irregular in extent, and vascularization is common though not invariable, presenting an appearance similar to that of inherited syphilis, without iritis, as opposed to Nuel,[1] who states that it is usually secondary to irido-choroiditis. Stephenson[2] finds the average period after infection is 10.8 years. Two-thirds of the cases were monocular. Only in two or three was the condition found within one year after infection. Distinctive features from the inherited were the monocular character, patchiness of the corneal changes, though most marked was the effect of treatment.

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[1] R. L. O. H., 1903.

[2] T. O. S., 1908.

There is a form of interstitial keratitis secondary to syphilitic irido-cyclitis. Together with the symptoms of an associated iritis there are small, round, grayish-yellow infiltrates in different layers of the cornea. In irido-cyclitis there is also an opacity in the cornea, triangular in outline with its apex upward which results from a collection of posterior corneal deposits.

Keratitis punctata syphilitica is an unusual disease first described by Mauthner. In the deeper layers of the cornea a number of grayish punctate infiltrates appear between which the corneal tissue often remains clear. The disease affects one or both eyes and causes very slight symptoms. The iris is involved, though not to the degree seen in irido-cyclitis. The opacities appear rapidly and under appropriate treatment usually disappear completely. The condition is observed in the secondary or in the tertiary stage.

Dimmer mentions two diseases of the cornea which have some relationship to lues. The first one of these, **keratomalacia,** is observed in children suffering from inherited syphilis in the early months of life. The disease shows itself with a superficial xerosis of the cornea and conjunctiva. The surface is dry, and is covered with a thin, white scum. This is associated with a rapidly developing gray opacity of the cornea which turns yellowish, and the cornea is often completely lost with but very few symptoms. In older children night-blindness can be observed. In two cases which have been reported by Peltesohn, general prostration was not noticeable. In other cases the children have a severe intestinal disorder and are in a very poor state of nutrition. The other form is **keratitis neuroparalytica,** which is the result of paralysis of the fifth nerve. It begins with epithelial desquamation over the entire corneal surface. The cornea then becomes dull and perforates with the usual sequelæ. In the mild cases the opacity of the cornea is the only change. The syphilitic processes which involve the fifth nerve are usually gumma at the base of the brain, meningitis or hemorrhage. In some cases other nerves are involved.

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[1] Norris and Oliver. System of Diseases of the Eye, Vol. IV.

[2] Ophthalmoscope, 1903, p. 170.

SCLERA.—Primary **scleritis** is an unusual condition; it is more frequently secondary to syphilitic disease of the uvea. The superficial form of scleritis shows a swelling of the sclera, which is bluish-red, on which the conjunctiva, with its brick-red vessels, is movable. An associated inflammation of the uvea may be absent. The **gumma** of the sclera is a form of scleritis in which the inflammatory symptoms are but little pronounced. There is a circumscribed reddening of the ocular conjunctiva with moderate subjective symptoms. A painless, sharply-defined tumor appears, which projects the conjunctiva before it. It may reach the size of a hazel-nut. This gumma may be the only one, or there may be two or three, and in some cases both eyes are affected. The most frequent site is the temporal and upper quadrant of the sclera. With appropriate treatment, the gumma disappears within two months; otherwise, the swelling breaks down and an ulcer develops in the middle of the tumor. There are usually other signs of tertiary syphilis. In literature there are a number of descriptions of gummata of the sclera—tumor-like, yellowish, painless thickenings occurring in the sclera underneath the dilated conjunctival and episcleral vessels. There are associated disturbances in the iris and in the vitreous. Prognosis is necessarily not bad, if appropria t treatment be instituted.

Iris and Ciliary Body.—Those affected with syphilis have **iritis** in a percentage varying between 0.8 and 6. In practically one-quarter of all cases of iritis syphilis is the cause. Gutmann, whose statistics on iritis are probably the most recent, found syphilis in 31 per cent. of the cases. The form of inflammation is usually a plastic one, and in the severe cases the ciliary body is also involved. That form of iritis known as iritis serosa, which is really a chronic cyclitis, is unusual in syphilis. Roseolar syphilide appears in the iris as distended vessels on the iris surface which are seen at the time of the exanthem. This injection of the vessels occurs particularly at the lesser iris circle. Papules are seen in the iris in a certain number of cases, and gummata occur in the iris as well as in the ciliary body. The symptoms of a plastic iritis in syphilis are similar to those in other forms of iritis. Hypopyon occurs in a small proportion of cases; more rarely there is blood in the anterior chamber. The gelatinous exudate is a grayish exudate which represents a cast of the anterior chamber, and at one stage suggests the presence of a lens dislocated in the anterior chamber. It contracts later, and disappears, often rapidly leaving a perfectly clear picture of the underlying structures. Fibrinous exudation is not characteristic of syphilis and occurs in a number of other forms of iritis.

Iritis usually begins in one eye. The second eye becomes affected in one-quarter of all the cases. In one-third of the cases the usual complications, of an iritis are present. There may be cyclitis (posterior corneal deposits); more frequently, in 30 per cent. of the cases, there are choroiditis, retinitis, interstitial keratitis or optic neuritis. In 6.5 per cent. of the cases corneal infiltrations are observed (interstitial keratitis). These differ from the ordinary interstitial keratitis in that the opacity is usually triangular and situated in the periphery of the cornea with no vascularization. In other cases it occurs in the form of a triangle, with the apex directed upwards, and is covered with a dull surface. This opacity is secondary to deposits on the posterior corneal surface. There is generally an exudation into the vitreous and into the posterior chamber. The former can be seen with the ophthalmoscope and explains the marked diminution of sight. An acute cyclitis which causes great pain is shown by œdema of the lids and tenderness on pressure.

In syphilitic iritis the inflammation of the iris may rapidly run its course and leave but a few adhesions. In the severe cases the pupil becomes occluded by a newly formed membrane, or secondary glaucoma results from seclusio pupillæ, and the pupillary part of the iris is shaped like a crater. Extensive exudation in the posterior chamber causes a flat adhesion of the iris to the lens, and the iris will then show the normal curve of the lens surface. This is followed by the development of a cataract (cataracta accreta). As a result of the exudation into the vitreous, the vitreous tissue may shrink, causing detachment of the retina with atrophy of the eyeball. Serous iritis (chronic cyclitis) is unusual in syphilis. It is characterized by slight exudation into the tissue of the iris, and the pupil reacts to the use of atropin. On the other hand, there are many deposits on the posterior corneal surface and the vitreous is clouded. The inflammatory symptoms are not marked. In the severe cases complications which result from an adhesion of the iris to the lens are absent, but the increasing vitreous opacity gives the greatest trouble. Complications in the deeper parts of the eye are more frequent. In a number of cases of irido-cyclitis, retinitis is found present. Relapses are frequently observed and probably depend upon a still existent general infection. Iritis, if uncomplicated, gives a good prognosis. When the ciliary body is involved and relapses occur with severe reduction of vision, it is serious. In this connection it is of interest to note how infrequently signs of preceding iritis are found in the eye lesions of tabes and paresis. The ordinary form of iritis is not characteristic if we disregard those very suspicious areas of circumscribed redness and swelling (roseola) which are sometimes observed. The presence of papules in the iris is however fairly characteristic and a diagnosis of syphilis may be made from them. Prognosis is always doubtful and worse than in the idiopathic form. In about one-half of the cases there is permanent damage to the sight.

The papules which occur in syphilitic iritis were formerly regarded as gummata, but as they occur in the secondary stage, it is better to speak of them as papules, leaving the gumma as a sign of the tertiary lesion, characterized by the absence of inflammatory symptoms. These papules have been found present in 8.85 per cent. of the cases. They may be present from the beginning, or occur during the course of the iritis. They are yellowish-gray or yellowish-red nodules, varying in size, covered with dilated vessels, usually situated at the pupillary, rarely at the ciliary margin. If they are situated in the former position a posterior adhesion is present. After they disappear they may leave no change in the iris tissue, or the iris atrophies at that point with a posterior adhesion. The papules may be so numerous about the lesser iris zone as to form a ridge at the pupillary border. The situation at the ciliary portion of the iris is less frequent. Sometimes the nodule in the iris tissue can only be suspected by a swelling and thickening of the iris tissue. Michel describes a form of syphilitic iritis where the region of the lesser arterial circle is chiefly affected, appearing swollen, of a dirty grayish-yellow color with one or more papules at the pupillary border.

In tuberculosis, the nodules are smaller and more frequent, more often situated in the ciliary border, grayish-white in color, and with comparatively little inflammatory reaction. The patients are often younger and show other symptoms of tuberculosis. In syphilis the nodules are fewer in number and are accompanied with more acute symptoms, are more apt to be situated near the pupillary margin, are of a more red color, and disappear with anti-syphilitic treatment. The patients are usually over twenty.

Gumma of the **iris** is a very unusual condition. It occurs singly, as a grayish-red tumor with enlarged blood vessels on its surface, frequently without inflammatory symptoms; it is larger than the papule, and is nearly always situated in the ciliary portion of the iris. After it has existed for some time, it will disappear by changing in color to yellow, leaving an extensive atrophy of the iris, or in certain cases it may rupture and fill the anterior chamber and even perforate the sclera. The cornea is frequently affected by an opacity of varying degrees, verging to a pronounced interstitial keratitis. If the gumma involves the sclera, it will cause a painful protrusion of this tissue which is bluish-black in color. Gummatous iritis occurs usually in one eye, and always late in the infection. According to Groenouw, papular iritis occurs somewhat earlier than the usual syphilitic iritis. The course of this form of iritis is more unfavorable than the simple iritis.

Iritis is rarely caused by **inherited syphilis** just as interstitial keratitis is rarely caused by acquired syphilis. Rabl found interstitial keratitis present in 34 per cent. of 127 children with inherited syphilis, and iritis in 3 per cent. While interstitial keratitis is a localization of late inherited syphilis, iritis occurs in cases where other symptoms of inherited syphilis appear in the first few months. In a few cases iritis was found present at birth. An acute iritis in the early months of life is always due to hereditary syphilis. The form is plastic; usually chronic rather than acute. The symptoms are so slight that it is only a discoloration of the iris which suggests the inflammation. There are in addition adhesions and closure of the pupil. Nodules are comparatively frequent. This form of iritis occurs either in one or both eyes. The complications are interstitial keratitis, vitreous opacities, choroiditis, neuro-retinitis and cataract. Under proper treatment a very fair amount of vision is frequently observed. The age at which the affection begins is usually at about the fifth or sixth month. The second period in which this inflammation seems to appear is between the tenth and eighteenth year. In two cases of irido-cyclitis due to inherited syphilis which I have observed, a zonular opacity of the cornea developed, notwithstanding, fair vision was obtained by operation. Brailey[1] described an iritis of the later stages of syphilis. This iritis occurs in adults thirteen years after infection, in the congenital form twenty-one years after birth (as opposed to the usual, where it occurs six months after infection or in infants six months after birth). This late iritis is usually double, an iritis serosa with tendency to secondary glaucoma; the type in the inherited is similar. Such an iritis may be accompanied with deep plaques in the posterior layers of the cornea, or appear as gummatous iritis with peripheric synechia, or be associated with a vascularizing interstitial keratitis. In the late iritis of inherited syphilis the general physical development of the patient is usually good.

Gumma of the **ciliary body** is either a primary condition or is an extension from a similar process in the iris. It is usually localized to one eye. If the eye has previously been healthy, there is pain and vision is diminished; then, after a few days, the sclera commences to bulge in the region of the ciliary body, and finally the iris is attacked. In other cases, a plastic iritis comes first and then, with a renewal of the inflammatory symptoms, a painful spot appears in the sclera. This usually is situated up and out from the cornea. The termination is generally unfavorable. The diagnosis is sometimes aided by the presence of other symptoms of syphilis.

Ewetzki[2] collected 67 cases of syphilomas of the ciliary body. They apparently occurred in the second stage in the form of papules, or in the tertiary stage as true gummata. It is only through the other symptoms and the general condition of the patient that a correct diagnosis can be made. Frugiuele distinguishes between benign and malignant forms of papules; the latter show a rapid tendency to extension leading to proliferation and shrinkage of the eyeball. True gummata occur in the tertiary stage. At the beginning the syphiloma of the ciliary body can not be identified and the case is a simple one of iridocyclitis. When the tumor has grown it may be seen through the pupil with the ophthalmoscope, or when it perforates. In 40 per cent. of the cases this perforation has taken place either externally or internally. A severe iridocyclitis is usually present with posterior deposits, hypopyon or hyphema. For diagnosis, it is important to remember that in sarcoma inflammatory symptoms are not probable and the rapid growth would also exclude a tumor. Tuberculosis must also be considered.

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[1] T. O. S., 1895.

[2] Ueber das Syphilom des Ciliarkörpers, Karger, Berlin, 1904.

Choroid.—The choroid is frequently affected in syphilis and the retina is affected with it, so that it is impossible to say which of these two members is primarily affected. It is, therefore, best to speak of chorio-retinitis. According to Groenouw, the statistics show that in 16.7 per cent. the choroid is involved and in 5.1 per cent. the retina is affected. The various forms of choroiditis which are seen in syphilis, with but one exception, are not characteristic. They are the following:

Acute Irido-choroiditis.—This means the extension of an iritis to the deeper structures as is shown by a change in the tension of the eye and a diminution of sight. It is always a severe process. The fundus is not visible on account of the vitreous opacities. The complications make the prognosis serious. There is a chronic form which is characterized by vitreous opacities in which the iritis is not marked; usually no foci are to be seen in the choroid on account of the vitreous opacities.

Disseminate chorio-retinitis occurs without involvement of the retinal vessels and of the disc. These exudates begin frequently in the periphery and then cover the entire fundus. They are at first indistinct with pigmentary changes in the margin and at the center, without the choroidal vessels becoming visible. Later on the areas become more sharply defined when surrounded with pigment and the sclera can be seen shining through between the choroidal vessels. The changes in the writer's experience are scattered over the fundus without any relation to the retinal tissues which show no abnormality. They consist in areas of choroidal atrophy of a map-like outline with irregular pigmentation and subsequent retinal connective-tissue formation. The new patches are recognized by an œdema, a hazy of the overlying retina and a distinct choroidal congestion with occasional small hemorrhages. Later the haze and the red color go, the outline becomes defined with the formation of pigment and the process ceases to be active. The supposition that 80 per cent. of disseminate choroiditis were due to syphilis is now known to be wrong. According to Alexander, only those cases are syphilitic where opacities occur in the posterior part of the vitreous. At the present time the Wassermann reaction is a great help in diagnosticating syphilis, though in this disease in only a proportion of the cases is a connection with lues probable. In the syphilitic form we find dust-like opacities in the posterior part of the vitreous, a complicating iritis or interstitial keratitis. The choroidal spots are in no way characteristic.

In the diffuse form of chorio-retinitis (to be described) after the disappearance of the retinal clouding, circumscribed foci of adhesive chorio-retinitis develop which may be multiple, spreading over the entire background, or remain solitary and then preferably in the macular region. Similar forms without preceding opacity, *i.e.*, primary disseminate or central chorio-retinitis, unquestionably occur in non-syphilitics. These conditions have nothing characteristic for syphilis, and the view formerly held, that most of these cases were due to syphilis, has been gradually abandoned.

In a number tuberculosis has been brought in etiological relation.

Igersheimer in 1910, showed with the aid of the Wassermann test that only a very small percentage of chorio-retinitis in adults was due to syphilis. A much larger proportion is due to syphilis in childhood or youth, particularly as regards the primary disseminate form. Very few observations show the development of primary chorio-retinitis in syphilis without the preceding retinal cloudiness and vitreous opacity.

Foerster has described an **areolar choroiditis,** in which whitish areas surrounded with delicate pigment are situated at the posterior pole of the eye; this choroiditis may be caused by different infections. True **gummata** of the eye ground are very unusual. The ophthalmoscope shows a white prominence, apparently starting from the choroid, and secondarily involving the retina. There are inflammatory symptoms such as vitreous opacities, iritis, optic neuritis, etc.

Diffuse Syphilitic Chorio-retinitis.—This is a characteristic syphilitic lesion which was first described by Jacobson and then particularly by Foerster. The disease occurs in one or both eyes and the patients are usually between thirty and forty years of age. It occurs in the late secondary or early tertiary stage not before the second year and other secondary lesions have preceded the condition. The course is chronic and relapses are frequent. A frequent complication is iritis which may precede or appear simultaneous with the chorio-retinitis. There are usually glandular swellings in the neck and, according to Foerster, skin eruptions are present in 50 per cent. of the cases. It has been stated that retinal hyperemia is frequently present in the secondary stage of syphilis. This has, however, not been confirmed. This diffuse chorio-retinitis is, in comparison with syphilitic iritis, one of the unusual manifestations. The dust-like vitreous opacities are an early sign, and can be best seen by dilated pupil with diminished illumination by using a strong convex glass behind a plane mirror. They frequently remain for many years. The optic nerve is blurred and red, the margins are ill-defined and the opacity of the nerve-head extends into the neighboring retinal tissue. This opacity often can be seen to follow the larger blood vessels in the form of delicate white radiating lines. In the region of the equator the eye ground is clear. Retinal hemorrhages are exceptional. The arterial walls do not present any visible changes. The veins are more congested than normal.

According to Foerster in about one-third of the cases, small circumscribed, decolorized areas are seen in the pigment epithelium in the early stage, particularly in the region of the macula or in the periphery. In some cases the condition is complicated with various forms of disseminate chorio-retinitis. These foci are situated in the periphery and may escape observation. In the macula in about one-third of the cases there are groups of red or white dots, sometimes grayish spots. These changes are often difficult to see until the vitreous opacities diminish, though their presence is suspected by the defect in the visual field. After recovery a large white scar sometimes remains. Similar changes have been observed in the periphery of the retina.

The subjective symptoms are of great importance. There is rapid diminution of vision which can not be explained by the changes to be found with the opthalmoscope. The visual field shows a more or less complete ring defect surrounding the central area. Sometimes there are isolated sector-like defects in the periphery. These make themselves noticeable to the patient by an area of scintillation. The ring-like defects are incomplete and more irregular than those found in retinitis pigmentosa. Their cause is not clear; some make the course of the blood vessels responsible. Another important symptom is night-blindness. The light sense is very much reduced. In bright illumination the patient's sight may not be very much affected. Furthermore, there are photopsias usually in the region of the defect in the field, which are increased on bodily exertion. Micropsia and metamorphopsia are also complained of.

The course is a slow one, and vision gradually diminishes. If early treatment is instituted, there may be complete recovery, or with partial defects. On the whole, the process is characterized by obstinacy and chronicity of course. The recovery frequently is incomplete and the disease goes into a chronic stage where even with prolonged treatment no particular improvement results. If the treatment is interrupted too soon relapses occur with further deterioration of vision. There is however a great tendency to relapses for which changes in the eye ground do not offer an explanation; there is then a further diminution of vision with a renewed onset of subjective symptoms, of photopsias, night-blindness, and defects in the field. Complete recovery occurs only in the mild cases and where no distinct defect in the field is present. Vision may improve for years, though it is generally somewhat impaired. In these cases vitreous opacities, choroidal atrophies, pigmentations, atrophy of the retina and various pigmentary changes are present.

If the process lasts for a long time, other symptoms regularly develop at the disc, the retinal vessels, the pigment epithelium and the choroid, and slowly the so-called chorioretinitis pigmentosa develops. The diffuse clouding of the retina and vitreous may last for years, before it ceases. In the severe cases the retina and the choroid undergo marked atrophic changes similar to retinitis pigmentosa. The pigment, however, is in the form of round deposits and not near the blood vessels. The optic nerve shows retinal atrophy. Opacities frequently form at the posterior pole of the lens. In the subsequent course of these cases, attacks of apoplexy or mental disturbances have been observed.

It is important to realize that if this process exists for a long time it can entirely change its appearance, and the diffuse retinal clouding is replaced by extensive atrophy of the pigment epithelium and multiple black pigment spots, or by circumscribed areas which are decolorized and exhibit pigment proliferation.

Certain symptoms observed in the first period suggest from the form of visual disturbance an early involvement of the external layers of the retina. The involvement of the choroid is still a mooted question. The ophthalmoscopic symptoms are explained by changes in the pigment epithelium and the presence of choroiditis in the later stages possibly indicates an extension of the disease from the retina to the choroid. Leber suggests that the name should be governed by the ophthalmoscopic picture, and when the retina is clouded we should speak of a retinitis, and when there are changes in the pigment epithelium with retinal pigmentation the condition is a chorio-retinitis.

There is a comparatively rare form of circumscribed infiltration which occurs either in the macular region as chorio-retinitis centralis, or in the periphery. This is not always to be distinguished from the diffuse chorio-retinitis just described, as in the latter, changes in the yellow spot may also be present. In the first group there are in the region of the macula circumscribed, pale, distinctly prominent areas, round and of considerable size, often several disc-diameters in size. About this large area there are sometimes smaller foci. The color is yellowish or grayish and there are a few retinal hemorrhages. If situated near the disc, the disc shows slight inflammatory changes. The vessels show no change. There may be vitreous opacities. The central location of the focus produces a central scotoma with marked diminution of vision. Improvement may follow treatment, though normal vision is not again obtained. The course is also in this variety tedious and prone to relapses. The condition fortunately is usually in only one eye. There may be an associated iritis. The patients are in the early stages of syphilis.

Hirschberg reported in 1888 a case of this kind as chorioretinitis tumida. In these cases there are probably small tumor-like proliferations of the choroid with secondary involvement of the retina representing an early stage of a gumma. A number of gummata of the retina have been described.

Retina.—As has been said, it is difficult to distinguish between diseases of the choroid and those of the retina, as these two tissues are so closely associated; yet it seems that there are lesions which principally affect the retina. In these cases the condition can be recognized with the ophthalmoscope as being situated in the retina, as sometimes having bearing to the course of the vessels and not leaving any pigmentary changes, but rather a connective-tissue scar (sclerosis). For purposes of description, it is convenient to speak of diseases of the retinal tissue (retinitis) and diseases of the retinal blood vessels. The former are much more frequent than the latter for the reason that as has been just stated, the retina is rarely primarily involved and is usually secondary to a choroidal process. Syphilitic diseases of the retina do not usually present the intense picture of an inflammation with pus formation. They occur in the form of a diffuse inflammatory clouding and swelling or a circumscribed cellular infiltration accompanied by a gradual degenerative process in the pigment epithelium. Most of the cases occur in the secondary period in which the diffuse syphilitic chorioretinitis is typical. The circumscribed cellular infiltrations are very much less common and are not found in any definite stage, though in the tertiary period they resemble a gumma. If the disease lasts a long time diffuse or circumscribed changes in the pigment epithelium result with secondary retinal pigmentation, which is the usual manifestation of hereditary syphilis.

Some writers (Jacobson, Alexander) speak of an independent syphilitic retinitis. While in the diffuse chorioretinitis the choroidal blood vessels are primarily affected with the result of causing changes in the choroid and in the external layers of the retina, it is possible for the syphilitic process to attack the retinal vessels and the resulting changes would then be in the internal layers of the retina. This differentiation in the writer's opinion is difficult to recognize with the ophthalmoscope, owing to the haze of the vitreous which occurs in both forms. In syphilitic retinitis there is a grayish cloud which hides the optic nerve and the surrounding retina, causing the disc to appear as a yellowish-red spot with blurred outlines. In addition there are hemorrhages, exudates, œdema and changes in the vessels; the last are very difficult and often impossible to see.

A **retinitis circumpapillaris** has been described in which the retina surrounding the disc up to the macula is swollen like a wall, while the disc itself is not affected. This condition differs from papillitis, in which the choroidal ring is hidden by the swelling of the nerve-fibre layer. The vitreous opacities are usually diffuse and consist of many dust-like dots which show rapid movement and sometimes seem to be deposited upon delicate membranes.

Hemorrhagic Form of Syphilitic Retinitis.—Though hemorrhages as a rule are absent in syphilitic retinitis, there are cases where hemorrhages occur in large number and justify the term of hemorrhagic inflammation. Occasionally these hemorrhages occur with distinct changes in the vessel walls.

Extensive hemorrhages sometimes give rise to preretinal connective-tissue proliferation, which produce bluish-white bands situated on the inner surface of the retina, causing a circumscribed detachment and are a mild form of proliferating retinitis. There are cases where, in the absence of vascular changes in the vessel walls, many hemorrhages are scattered over the retina. This disease was described by Schoebel in 1898. It is usually bilateral, with marked inflammatory symptoms. Opacities of the vitreous are present, and, in addition, there may be profuse hemorrhages into the vitreous which take a long time for their absorption and often cause detachment of the retina and vessel formation in the vitreous. These profuse vitreous hemorrhages in syphilis cause a black background to the ophthalmoscope, though blood can sometimes be directly observed by oblique illumination. They occur late in the infection in eyes which have been affected with chorio-retinitis and with vascular disease.

Central recurring retinitis is a very unusual type of inflammation which has been observed in the late periods of syphilis. The visual disturbance is characteristic and consists in the sudden onset of marked central loss of sight which, after a few days, passes off and returns after a number of weeks or months. These attacks have been known to recur from thirty to eighty times. Treatment has but little influence. As this condition occurs in the late manifestations, other changes are usually present. The scotoma may be in the form of a ring, or there may be micropsia. Ophthalmoscopic changes are slight in proportion to the disturbed vision. The delicate clouding of the retina in the region of the macula of a grayish color is observed sometimes in combination with small white spots and a few hemorrhages. The changes are not easily to be seen on account of the vitreous opacities. Between the attacks the ophthalmoscopic picture is normal. Later on a permanent opacity develops with some pigmentary changes. During the attacks there is some photophobia and ciliary congestion. It occurs in both eyes or in only one eye. The attacks gradually cease and the amount of vision depends on the permanency of the retinal changes. Leber is inclined to believe that the condition depends upon a shallow circumscribed detachment of the retina.

Retinal Vessels.[1]—Changes in the vessels in the various forms of syphilitic retinitis are to be separated from the more or less independent changes of the larger vessels where there is a clouding of the vascular wall without ophthalmoscopic evidence of a retinitis. In the diffuse chorio-retinitis of the secondary stage no changes can be seen in the retinal vessels. In the form of retinitis which occurs in the later stages, characterized by changes in the pigment epithelium and retinal pigmentation the so-called chorio-retinitis pigmentosa, the vascular wall is opaque and thickened with corresponding narrowing of the lumen. The vessels are accompanied by white lines and become thinner as they approach the periphery, and finally disappear entirely from view. In the advanced cases the vessels on the disc are very thin and can only be followed for a short distance in the retina.

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[1] Leber, Graefe-Saemisch-Hess, II ed., 1915.

Independent vascular disease of the retina of syphilitic origin is unusual. It occurs in the late periods of syphilis usually without any signs of retinitis, in one or in both eyes, with a pronounced clouding of the vessel wall with only a slight narrowing of the lumen. It develops rapidly and may completely disappear. It occurs generally in the arteries, occasionally it has been observed in the veins or in both vessels. Syphilitic arteritis was first described by Haab and he has published the picture of a very striking case. The opacity of the vessel wall is usually marked. The blood column is narrowed, surrounded by broad white lines or completely covered. Branches or a large subdivision of the blood vessel are involved; rarely there is a uniform distribution over the entire vascular system. The circulation is preserved so that there is no marked diminution in the function. In the particular segment of the retina affected numerous hemorrhages may be present. These changes in the vessel wall may be entirely cured with energetic treatment. In a case of the writer's where the course of the syphilis was curiously hastened by the exclusive treatment with salvarsan, the superior temporal retinal artery became surrounded by an exudate like an injected lymph sheath and a few hemorrhages. This vessel became obliterated and a permanent sector-like defect in the field resulted. The eye showed other syphilitic manifestations:

iritis, vitreous opacities, choroiditis. A very remarkable condition has been observed by de Schweinitz[1] where the process affected all the blood vessels of the eye together with retinal hemorrhages and which Leber regards as of syphilitic origin.

Vascular changes of this character have been observed in cases where syphilis has produced a nephritis. There have been cases where symptoms of cerebral lues were present. Some authors believe that the demonstration of these retinal changes is of importance for the diagnosis of brain syphilis. Uhthoff, however, was not able to substantiate the frequency of these vascular changes, and found that pronounced ophthalmoscopic retinal lesions are practically absent in syphilis of the cerebral nervous system. This view is confirmed by a study of Geis's material, in which there were many cases of cerebral lues, but syphilitic changes of the retinal arteries and hemorrhages were very unusual. Three patients alone could be followed longer than from four to six years. Two of these were well and showed no symptoms of cerebral disease; the other died after three years, from a cardiac lesion. Syphilitic disease of the retinal arteries does not have the same prognostic importance which corresponding arterio-sclerotic changes in the retina possess.

Chorio-retinitis in Hereditary Syphilis.—In hereditary syphilis during the suckling period a bilateral affection of the eye grounds has been observed which may belong to the type of **chorio-retinitis** or to neuro-retinitis, in which the vitreous is also involved. The disease is apt to be overlooked unless nystagmus, strabismus or improper holding of the head draws attention to a possible disturbance of the eyes. Hirschberg, in 1886, described a disease of the eye grounds occurring in syphilitic children between the fifth and eighteenth months, which is regularly bilateral. The disc is clouded, grayish-red or sometimes presents a bluish-white exudation which extends for a part into the retina. This disappears after a few weeks and may leave some connective-tissue formation on the disc or changes in its color and contour as well as narrowing of the arteries. Throughout the eye ground there are numerous pale dots which are crowded together in the periphery with pigmentation. Sometimes the choroidal areas are large and red or white in color with pigmentation which somewhat resembles the typical pigment degeneration. In the macular region there is a dark brown discoloration or a grayish-black area.

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[1] de Schweinitz, T. A. O. S., 1907.

The writer has observed a similar fundus change in a child aged four and one-half, with a strongly positive Wassermann, who was brought to the hospital because the mother had noted loss of sight. The retina was cloudy, the disc was bluish-white and blurred, there were some bright choroidal atrophies, while in the periphery diffuse chorio-retinal changes were present. With treatment marked improvement was obtained, the nerve-head became dirty gray and the arteries were thin.

Observations from the children's clinics in sucklings with hereditary syphilis have shown an unusual frequency of **papillitis.** The media were clear, the discs swollen, the margins blurred, and occasionally there were small hemorrhages. These changes all disappeared under treatment and the discs resumed their normal color. Exceptionally they became atrophic; choroidal changes and vitreous opacities were sometimes present. This observation is noteworthy as in later years changes of this character are not observed; possibly the great mortality of these cases must be taken into consideration. In any case, it shows that an early ophthalmoscopic examination in doubtful cases is of value for the diagnosis of congenital syphilis.

Another form of **chorio-retinitis** is that occurring in conjunction with parenchymatous keratitis. This is present sometimes at the onset of the keratitis and may have developed a long time before. In other cases it is not recognized until the cornea has cleared.

These forms are in general chronic with only slight retinal clouding or hyperemia, and Leber regards them as degenerative chorio-retinal processes. The affection occupies chiefly the periphery of the eye ground and a number of pictures are presented according to the amount of pigmentation and the size of the atrophic areas. The peripheric location does not produce visual disturbances, consequently its onset is not observed. It may occur independent of parenchymatous keratitis, and not be noted until the patient is about twenty and a complicating glaucoma arises.

The changes in the eye grounds which occur in inherited syphilis show many different forms. At the same time the attempt to classify[1] them is more or less successful. The first type is represented, according to Haab, by diffuse small spotted changes which give the eye ground the appearance of having been covered with pepper and salt, which may involve the periphery or the entire fundus.

A similar condition is found in tapeto-retinal amaurosis (Leber) which is not due to inherited syphilis, and occasionally in the so-called pigment degeneration without pigment. The optic nerve and the vessels may be normal. In other cases the nerve-head is pale and the vessels are narrow. The vitreous is clear, the pigment spots gradually enlarge, become angular and change to the fourth type, as has been observed in a number of cases. This form is always bilateral, occurs early in life (the seventh week to the fourteenth month). Leber believes that it is a later stage of the previously described infantile papillo-retinitis, where the changes of the disc, in the retina and in the vitreous disappear.

The second and third types are characterized by the appearance of larger round chorio-retinal areas which vary in size, in which the foci are discrete or crowded together, with resulting irregular lobulated areas. The periphery of the eye ground is especially affected. The vitreous shows circumscribed opacities. The optic nerve and the retinal vessels are not particularly changed and vision is good. In the second group the areas are pigmented; in the third the atrophic areas have but little pigment.

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[1] Sidler-Huguenin, Ueber die hereditär-syphilitischen Augenhintergrundsveränderungen, etc., Leipzig, L. Voss, 1902.

Autonelli, Les stygmates ophthalmoscopiques rudimentaires, etc., Paris, Maloine, 1897.

Hirschberg, D. m. W., 1895, Nos. 26 and 27.

Haab, Atlas. München Lehmann. In English, Philadelphia, Saunders.

Silex[1] has described in individuals under fifteen years a form which he calls choroiditis areolaris, in which there are black areas with a bright center, or pale areas with a black margin, particularly in the macular region. In this form vision is affected and the condition may be limited to one eye.

The fourth type includes cases in which there is a secondary pigmentation of the internal layers of the retina, optic atrophy, narrowing of the blood vessels, a picture resembling pigment degeneration. In addition to these pigmentary changes, there are large chorio-retinal areas which may occupy either the periphery or the posterior part of the fundus. The main difference between this condition and true pigmentary degeneration is that there is no distinct localization of the pigment near the visible blood vessels.

These types may be combined. Types 2 and 3 are closely related to interstitial keratitis which they precede or rarely follow.

Pigmentation about the disc is observed in other conditions, and is not pathognomonic, as opposed to Antonelli's view.

The changes in the eye ground which depend on hereditary syphilis may be only very slight and the process remains rudimentary. In unusual cases the changes may be so extensive as to lead to complete blindness. In a few cases the process has caused a detachment with complete destruction of the eye. Nettleship has reported a number of these cases which resembled glioma of the retina.

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[1] Silex, B. kl. W., 1896.

There are a number of observations which prove that syphilis may be transmitted to the third generation (Leber) where it has caused chorio-retinal processes, chorio-retinitis, areolaris and pigmentosa, and interstitial keratitis.

Collins[1] examined the children of 12 women with inherited syphilis and found them free from taint, which he regards as presumptive evidence against the transmission of syphilis to the third generation. The mortality of these children is unquestionably greater and a dystrophic influence is inferred. Sidler-Huguenin[2] has not observed eye changes characteristic of inherited syphilis in the third generation and find the children rickety, tuberculous or backward mentally and physically.

Igersheimer,[3] in an interesting study of the future history of interstitial keratitis cases, found that of 152 eyes good vision was obtained in 59 per cent. of the cases. In 44, myopia developed; in 5 cases amaurosis resulted. Prognostically the age of the patient at which the disease manifests itself is of importance. The younger the patient, the better are his chances. In most cases the second eye is as severely affected as the first. The tension was increased in 27.4 per cent.; a decrease was observed in 21.6 per cent. The ectatic condition of the cornea which could be taken as a result of increase of tension, was often associated with normal or even sub-normal tension. Relapses occurred in 14 per cent. of the cases. It is known that these patients suffer from other manifestations of inherited syphilis which shows itself in affections of the bones, teeth, the hearing, etc. An important complication has sometimes been overlooked, and that is the nervous system. This author found in not less than 43.5 per cent. severe forms of nervous disease. This is another reason for advocating the continuance of the anti-syphilitic treatment even after the eye process has come to a standstill. As to the offspring of these patients, of 28 children the Wassermann reaction was positive in 12; in one case lues was present in the third generation. The author's statistics show that congenital lues does not prevent a new infection.

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[1] Collins, R. L. O. H. 1903.

[2] L. c.

[3] Igersheimer, Das Schicksal von Patienten mit Keratitis Parenchymatosa auf Hereditar-luetischer Grundlage. (Sammlung zwangloser Abhandlungen aus dem Gebiete der Augenheilkunde, Vossius, 914.)

Optic Nerve.—According to Groenouw, of all the inflammations of the optic nerve, about 18 per cent. depend upon syphilis. Primary syphilitic disease of the optic nerve is always inflammatory, while the simple atrophy is always secondary.

While Ole Bull[1] reported hyperemia of the optic nerve as one of the most frequent disturbances in early syphilis, this observation was not confirmed by later investigators. Krueckmann found hyperemia in only 3 per cent. of the cases. After the subsidence of iritis, ophthalmoscopic examination or the discovery of impaired vision will lead to the recognition of an optic neuritis which is not an infrequent complication. The outline of the disc is blurred and there is a venous congestion. The condition is obstinate but the prognosis with proper treatment is good.

There can be no question but that there is a **primary specific optic neuritis** where disease of the brain or of the orbit can be excluded. Primary optic neuritis may appear as a simple optic neuritis, a choked disc, or a retrobulbar neuritis either in one eye or in both. The field often shows a central scotoma, an annular defect or a peripheric defect. The ophthalmoscopic changes vary according to the proximity of the inflammatory focus. The surrounding retina is frequently affected and the process is then more properly spoken of as a neuro-retinitis. There are often other ocular manifestations of syphilis, such as iritis, choroiditis and vitreous opacities. The process occurs in the late secondary period and is very rebellious to treatment; a certain amount of visual defect usually remains.

Primary optic atrophy can occur as the first symptom of tabes and paresis and precede the other symptoms by many years. There is no primary syphilitic optic atrophy, though the optic nerve is frequently involved secondarily in brain lues. In this we find optic neuritis, choked disc and simple optic atrophy (descending atrophy). These optic-nerve conditions are treated fully in the first half of the book.

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[1] Ole Bull, The Ophthalmoscope and Lues, Christiania, 1884.

An unusual condition is a gummatous proliferation of the optic nerve-head. A case of this character was reported by Scheidemann[1] while affections of the optic nerve back of the papilla have been reported by a number of observers. Leber speaks of the regularly present necrosis of the infiltrated areas of the optic nerve.

Orbit.—The orbit is affected in syphilis in the form of periostitis which is usually a tertiary lesion. It is unusual, according to Groenouw 70 cases have been reported. Notwithstanding the frequency with which syphilis attacks the periosteum of the cranial bones, the frontal and parietal in particular, the orbital walls are but rarely affected. Where the orbit is affected, other parts of the skull show similar changes. The favored site is the upper orbital margin and a hard tumor forms at a part of the orbital margin. After it has existed for some time osteophytic deposits form at the periphery, the tumor softens, the skin turns red and perforates, permitting the escape of purulent contents; a fistula results leading down to carious bone. In the subsequent cicatrication the eye lids sometimes become deformed.

The periostitis may select a different site at any of the orbital walls. The symptoms then in addition to pain consist in exophthalmos. When situated at the apex of the orbit, there will be exophthalmos, swollen lids, pain, a paralysis of the various motor and sensory nerves. The V nerve is sometimes affected so that there is associated anæsthesia, which may lead to neuro-paralytic keratitis. In most cases other ocular nerves are involved including the optic nerve. The optic nerve may remain healthy, or become affected in the form of optic neuritis with hemorrhages, which usually results in a neuritic atrophy. Iritis has been mentioned as a complication and there are sometimes affections of the nasal sinuses. If the treatment is started promptly, the prognosis is good.

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[1] Scheidemann, C. f. prakt. A., 1895

The lacrymal gland is rarely the seat of a syphilitic lesion, though a number of cases have been recorded. The condition is unilateral, occurring in the secondary or tertiary period. The gland enlarges, without pain, like a tumor, and the upper lid is somewhat swollen. Palpation reveals a hard tumor in the region of the lacrymal gland. In one case of the writer's the symptoms were those of a tumor in a woman of fifty; after removal the microscopic examination showed that the process was syphilitic. Other syphilitic eye symptoms are sometimes present. In a case of de Lapersonne multiple glandular swellings were present scattered over the body, in the testicle, mammary gland, and parotid. It is evident that the diagnosis is very difficult. The **lacrymal passages** are more frequently involved. Terrien describes a gumma of the lacrymal sac, a condition which has been observed in four cases. The tear passages usually suffer from a periostitis of the superior maxilla which obstructs the lacrymal duct; and in other cases the lacrymal sac is directly invaded from the disease of the surrounding bone. Associated with this are various lesions in the nose, ozena, necrosis, perforated septum, saddle-shaped nose, etc. Disease of the superior maxilla is particularly frequent in inherited syphilis and is the cause for dacryocystitis, lacrymal abscesses, and fistulæ in children.

MUSCLES.—Paralyses of the ocular nerves, including the facial and the fifth represent according to Groenouw about 18 per cent. of ocular syphilis. The third nerve was involved in 73 per cent., the sixth in 24, the fourth in only 3 per cent.; while the fifth nerve was affected in 2 per cent. and the facial nerve in 3 per cent. According to various authors ocular-muscle paralyses are due to syphilis in from 17 to 60 per cent. The cause of the ocular paralysis is usually an affection of the nerves, and the nerves are generally not primarily affected, but are involved secondarily by extension from the surrounding tissues. At the same time a primary syphilitic neuritis can occur in the optic nerve, and probably in the other cranial nerves, though the diagnosis can not be made. The most frequent cause for the ocular paralysis is a compression of the nerves through a syphilitic affection in the orbit, in the bony canals of the skull, or along the base of the brain. Nuclear and cortical disease are occasionally present following circulatory disturbances from syphilitic vascular disease. The ocular paralyses which are precursors to tabes and paresis do not belong to this chapter. In cerebral syphilis there are usually other paralyses and cerebral symptoms. An isolated paralysis of a single nerve even without other symptoms of a brain lesion does not exclude the possibility of a brain affection.

The third nerve is affected in 76 per cent. of the cases where the outer or both groups of muscles are involved, while in 24 per cent. of the cases the internal muscles are affected.

Isolated paralytic ptosis is frequent, though not characteristic for syphilis. It is usually of nuclear origin. If both third nerves are involved, the brain is then affected in its base between the two crura. Disturbances of the pupil and of the accommodation are frequently syphilitic, particularly the one-sided internal ophthalmoplegia, a reflex iridoplegia, which suggest lues. Paralysis of the fifth nerve may cause anæsthesia of the cornea and neuro-paralytic keratitis. Irritation of this nerve produces neuralgia, not infrequent in the early stage of syphilis, when it may be associated with paralysis of other muscles. Paralysis of a number of different ocular muscles is very frequent in brain disease. Conjugate deviation and external ophthalmoplegia is nearly exclusively caused by syphilis. Nystagmus may be produced by syphilis, secondary to syphilitic disease of the brain or cord. The ocular muscles are usually involved late, though there are a number of cases on record where a few months after the infection ocular paralyses have been observed. In this connection it is of interest to note the more frequent observation of ocular paralyses since salvarsan has been given. This subject has been treated in connection with neuro-recidives.

TUBERCULOSIS[1]

Occurrence—The eye becomes infected in tuberculosis either directly or more generally indirectly. A direct infection takes place in certain forms of conjunctival and corneal tuberculosis. A tuberculosis of the skin of the face (lupus) may extend and involve the eye lids, conjunctiva and the lacrymal passages, or the last may be affected by an extension of a tuberculosis of the nasal mucous membrane or of the neighboring bony structure. In the majority of cases the infection, however, is indirect, *i.e.*, secondary to some focus in the body. Manifest tuberculous lesions in the body are usually not present in cases of ocular tuberculosis, and eye complications in active pulmonary tuberculosis are very rare. I have seen an irido-cyclitis, without any characteristic eye symptoms in one, and attacks of fleeting episcleritis in two other cases where the lungs were actively affected. Experiments have shown that a tuberculous affection of the eye can take place through the intact intestinal mucosa (Calmette). The eye patients do not present any of the active symptoms that go with the popular conception of tuberculosis, and cases of eye tuberculosis are quite different from the pulmonary cases seen in the medical clinic.

The patients with ocular tuberculosis may give a suspicious family history; the personal history is often suggestible, occasionally there is a history of phlyctenular trouble in early life; on the other hand, at the time of the eye trouble they may appear to be in blooming health. The eye trouble is, however, a chronic one which does not yield to the usual treatment.

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[1] Groenouw, Graefe-Saemisch, II ed., 1904.

The frequency of ocular tuberculosis though suggested by Michel years ago, was not confirmed until after the general employment of tuberculin for diagnosis was established and some experimental work was done. The scientific proof for the prevalency of ocular tuberculosis was brought by Stock, who produced experimentally iritis, choroiditis, scleritis, etc. Leber, in 1891, suggested the concept "attenuated tuberculosis" as the cause for nodular iritis, a form which is capable of spontaneous recovery.

Every chronic eye trouble is suspicious, and if other causes can be excluded, tuberculosis must be considered. The diagnosis of a tuberculous eye lesion in many cases can not be made with certainty from the clinical picture. Heine[1] says that there are two ways of estimating the tuberculous nature of an eye lesion: (1) clinical examination of the eye and general examination of the patient including family and personal histories; (2) the specific reaction to tuberculin. Otherwise the diagnosis can only be made by the demonstration of the tubercle bacillus, microscopic examination of the tissue or by animal experiment. These methods are, of course, in most cases not available; of these the most definite is animal inoculation.

Inoculation Tuberculosis.—Fluid material is injected into the anterior chamber of a rabbit by means of a hypodermic syringe or particles of tissue are introduced in the anterior chamber. A mixed infection may cause suppuration of the eye, otherwise the material inoculated remains situated on the iris, is encapsulated, gradually shrinks and disappears. Usually after three to six weeks in the case of rabbits small grayish nodules appear in the iris, which increase in size and in number. The iris becomes swollen and congested, the cornea clouds, hypopyon develops, the proliferating mass finally perforates the eye ball, or the process may go on to absorption.

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[1] Heine, Erfahrungen und Gedanken über Tuberkulose and Tuberkulin, Medizinische Klinik, 1912.

A brief experience with tuberculin will convince every observer of its great value, and Schieck[1] does not exaggerate when he says that tuberculous eye diseases were first fully understood after the use of tuberculin. It must be insisted upon that the use of tuberculin, especially in the class of cases seen in ophthalmology, if followed out according to definite rules, and with certain restrictions, has never injured the patient. Tuberculin, while it has no effect in the healthy body, in the tuberculous even in small doses produces characteristic reactions. The preparation usually used for diagnostic purposes, known as old tuberculin, is a sterilized extract of a pure culture of the tubercle bacillus in glycerine. There are two methods by which it is applied.

Tuberculin Test.—1. Von Pirquet's **cutaneous** reaction results in an inflammatory reaction at the site of application. On the surface of the forearm a drop of the old tuberculin is applied to two areas about 4 cm. apart. The skin is superficially scratched without drawing blood at a point midway between these two places and at each of these places. After twenty-four to forty-eight hours a positive reaction shows itself by hyperemia and exudation and the development of a papule. This reaction is distinctly specific and indicates that the individual at some time or other has been infected with tuberculosis. Its value, therefore, decreases with the age of the patient. It is, in fact, too delicate, and a positive test is not regarded of value except in young children.

A much more important test is the **subcutaneous** one. With this, three reactions can be distinguished; general, arm (site of injection) and focal (in this case, the eye). The test consists in the subcutaneous injection of Koch's old tuberculin in 1, 3 and 5 *mg* strength; children receive ½ *mg*. A favorable site for injection is the arm. It is an absolute rule that the patient must be afebrile; it must be noted that the temperature does not rise above normal for two days before the injection. The temperature should then be taken every two or three hours. A strikingly subnormal temperature is not infrequent in these cases. The patients are to be kept quiet and in bed if the reaction begins to show itself. After twelve to twenty-four hours, sometime on the second day, in the positive cases a febrile reaction of from 1° to 3° ensues. The temperature rises abruptly, then gradually decreases by lysis. This is the typical febrile reaction. If after forty-eight to seventy-two hours there has been no rise in temperature 3 *mg* is given; then, if necessary, after the same length of time 5 *mg*. More patients react after the second than after the first injection. If there is no reaction after the third (5 *mg*) the condition is regarded as not tuberculous. Associated with the rise in temperature there are other important symptoms, accentuation of the pulse, chill, headache, pain in the extremities, backache, and general malaise. These symptoms generally last but one day.

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[1] Schieck, Die Immunitätsforschung im Dienste d. Augenheilk, Wiesbaden. 1914.

Bandelier and Röpke[1] give the following contra-indications to the diagnostic tuberculin test: (1) temperature over 37.3°C.; (2) positive previous history, clinically present physical signs and presence of bacteria; (3) hemoptysis; (4) heart disease; (5) nephritis; (6) epilepsy.

The arm reaction is a percutaneous reaction at the site of injection; this area may become red, indurated and painful. Hummelsheim finds that the intensity of the arm reaction corresponds to the height of the fever and regards it even as a more delicate test.

Together with the general reaction there is sometimes a focal reaction. The importance of this reaction is evident, as a positive general reaction does not result from a focus restricted to the eye, and unless the eye shows some change the proof of the tuberculous etiology of the eye lesion is not given. The changes in the eye are, however, often difficult to recognize, as they may be but little marked and may last only for a few hours. To detect these changes the eye has to be carefully examined twice a day under the best circumstances (artificial light and the loupe) and intraocular changes require functional tests and examinations with the ophthalmoscope. Heine does not insist upon the presence of a local reaction, as it is so often absent in unquestionably tuberculous eye lesions (iritic nodules) and believes that the absence of a local reaction in ophthalmology does not prove that the eye lesion is not tuberculous.

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[1] Bandelier und Roepke, Lehrbuch d. Spezifischen Diagnostik und Therapie der Tuberkulose, Würzburg.

Increased ciliary congestion, new foci in cornea, iris or episclera, increased haze of aqueous or vitreous, new hemorrhages and the appearance of small white dots about a process in the choroid or retina, are all evidences of a local reaction. After a positive reaction the congestion often becomes much less, the eye is whiter than it has been for some time, the vitreous is clearer and sight is improved. Heine calls this the negative local reaction.

Schieck[1] describes the following ocular changes in a positive local reaction. **Conjunctivitis:** no changes. **Keratitis:** increased ciliary congestion, epithelium clouds over suspicious opacities. The eruption of phlyctenules of infiltrations is not of definite importance. **Iritis:** increased ciliary congestion, clouding of aqueous, blood vessels in iris become more distinct. Schieck has never observed the formation of synechia or the dissemination of the tuberculous process after the diagnostic test. **Intraocular lesions:** moderate retinal haze over a recent choroidal process. Hemorrhages and vitreous opacities were not observed.

It must not be forgotten that the local reaction depends on an augmentation of the local inflammatory changes which exceptionally might produce undesirable symptoms, as in vascular disease of the retinal veins. Oloff[1] states that tuberculin is contra-indicated in eye cases where there is a tendency to hemorrhage. In retinal lesions small hemorrhages and small round whitish infiltrates may appear in the periphery of a focus after a general reaction to tuberculin. In a case[2] of the writer's a crop of tubercle-like infiltrates appeared about a retinal focus near the optic disc; these subsequently disappeared without leaving any trace.

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[1] L. c., p. 77.

Tuberculin treatment in its modern application is the work of A. v. Hippel. It consists in a form of active immunization, a stimulation of the body's natural protective resources, and depends generally speaking on the use of infinitesimal doses and the avoidance of the slightest local or general reaction. The injections should be given at definite intervals (twice a week, then once a week, etc.). A rapid cure is not possible, and when these are reported the diagnosis was probably incorrect. It demands great patience on the part both of the patient and of the physician, and definite individualization. The form of tuberculin now generally used in treatment is the bacillary emulsion. The value of the treatment is not accepted by all, especially as in some forms its action is disappointing. At the same time, excellent results can be obtained in some cases if the treatment is persevered in. Relapses of course occur. Another question which is unsolved, is when should the patient be regarded as cured? As far as the eye is concerned this would mean the arrest of the ocular process, the complete cicatrization of foci and clearing up of exudates. Should the general condition, the hidden focus in the body, then be further treated? Petrushky[3] waits for three months after an apparent cure to find whether a renewed sensitiveness to tuberculin has appeared; in that case treatment is carried out through another stage even without any clinical symptoms of relapse.

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[1] M. m. W., Mar. 31, 1914.

[2] Published with picture in the Arch. of Ophth., 1913.

[3] Petrushky, Grundriss d. specif. Diagnostik und Therapie der Tuberculose, 1913.

Our results have been best in episcleritis and in retinal lesions.

Heine gives a statistics of 125 cases where in half a favorable influence was noticed. Relapses occurred in 11 after completion of treatment—good results were distinctly influenced by the earliness at which treatment was begun.

Forms of Ocular Tuberculosis.—Eyelids.—Tuberculous lesions of the eyelids are very unusual in this country. They show themselves in the form of lupus of the skin, in the form of a chalazion, or infrequently as tuberculosis of the lid cartilage. The swelling of the neighboring gland is of importance in the diagnosis, though the demonstration of bacilli is essential. Lupus is characterized by small nodules of varying size of brownish-red color, of transparent appearance, and of soft consistency. The course is chronic, the tissue is destroyed, ulcerates, and forms scars, these terminating in deformity of the lid. Lupus is usually transmitted to the eyelids from a similar affection on the face. Some have claimed that the ordinary chalazion is tuberculous. The presence of giant cells, however, are only characteristic of a foreign-body tuberculosis.

Conjunctiva.—Tuberculosis of the conjunctiva usually occurs in the palpebral portion, especially the upper, and then extends to the retro-tarsal folds. It may begin in the latter situation or rarely on the ocular conjunctiva and in the semilunar fold. The disease is often limited to one eye lid, which externally shows an increase in size and resistance with increased secretion.

The diseased conjunctiva presents a peculiar partial discoloration in addition to marked congestion. The conjunctiva is transformed into granulation tissue which may ulcerate and be covered with a gray membrane, and in places shows a whitish gray or a dirty red color. The parts surrounding these ulcerations present irregular hypertrophies which may contain gray nodules. The swollen conjunctiva may, on the other hand, present numerous round and oval structures of a grayish-yellow color which resemble follicles and apparently show but little tendency to necrosis. Michel has stated that the clinical picture of trachoma can be produced by a tuberculous infection. There are cases where no ulcers form and the tuberculous lesion shows itself by numerous small gelatinous nodules situated in the depth of the tissue. Rarely the process presents a circumscribed polypoid prominence covered with smooth tissue, or the tarsus and the intermarginal part of the lid may be involved. The margin of the lid then looks as if it had been ground down, the eye lashes are lost and the adjoining part of the skin shows fine scars.

Sattler has described four principal forms: First, that which resembles the usual tuberculosis of the mucous membranes in other parts of the body. Second, there are numerous small grayish nodules like trachoma follicles with very slight tendency to necrosis and signs of caseation. These occur in the retro-tarsal folds, in the semilunar fold and in the ocular conjunctiva. In the third variety, the proliferation in the palpebral conjunctiva is more marked with pronounced mucopurulent discharge. The surface of the conjunctiva looks like a mass of exuberant granulations. The fourth variety is lupus of the conjunctiva. Eyre has added a fifth, in which he described a tuberculous hypertrophy which resembles a pedunculated tumor like a papilloma, situated on the tarsal conjunctiva.

The cornea is not infrequently affected, usually in the form of pannus. There is superficial opacity and vascularization with irregularity of the epithelium, and occasionally nodular prominences. Deep inflammatory processes, ulcerations and infiltrations are rare; though the lacrymal sac may be involved in these tuberculous conditions. Aside from the lesion in the conjunctiva, tuberculous processes may be situated about the nasal openings and the palpebral fissure. In these cases which are called lupus of the conjunctiva, the symptoms are somewhat different; the papillary hypertrophy is not so pronounced and there is a greater tendency to the formation of scars and an involvement of the margin of the lids and of the tear passages.

In other cases the conjunctiva is involved by the extension of a tuberculous lesion of the lids or of the nasal mucosa. Lacrymal passages become affected usually secondarily to a diseased nasal mucosa or to a tuberculosis of the adjoining bony structures. Thus Hessberg found in 8 cases of tuberculosis of the nasal mucous membrane an infection of the lacrymal passages in 5. Involvement of the lacrymal passages present the clinical picture of a blennorrhea of the sac or of a lacrymal fistula.

The early involvement of the lymphatic glands, particularly the preauricular gland, is very characteristic for tuberculosis of the conjunctiva. This is so constant a symptom that it is of value in the differential diagnosis.

The course is always chronic and at first there are but few symptoms. The patient's attention is drawn to his condition on account of the heaviness of the lid, the increased discharge, and the corneal complication. The disease is usually one-sided. Only in 12 out of 97 cases, according to Saemisch, were both eyes involved. In lupus, however, the condition is more frequently bilateral. This affection usually attacks young persons between ten and thirty years of age. The microscope reveals a vascular granulation tissue containing numerous round nodules which undergo necrosis in the form of caseation.

The diagnosis is difficult from the clinical symptoms. It can only be confirmed by the demonstration of tubercle bacilli. As these bacilli are rarely to be found in the discharge, the only definite method is animal inoculation. The conjunctival lesion is a primary direct infection or an extension from the skin, lacrymal passages, or bone, though it may be metastatic. The use of tuberculin for diagnostic purposes is not definite. The disease may be mistaken for trachoma, especially those cases in which the tuberculous process does not cause ulceration, and there are follicle-like nodules deep in the tissues. A pannus is common to both diseases. On the other hand, in tuberculosis ulceration of the conjunctiva is very frequently present and the disease affects usually only one eye.

Tuberculosis of the conjunctiva in the large majority of the cases is a primary disease depending upon an ectogenic infection. In most of the patients other signs of tuberculosis are absent. Preceding injury has been found to facilitate the infection of the conjunctiva. Thus, it has been observed after injury from small foreign bodies, after an operation for squint, etc.

Prognosis is extremely grave, though it is possible for the disease to be a self-limited one. If left to itself, the process extends along its surface as well as into the depth. It can invade the tarsus, the fornix, the ocular conjunctiva, the cornea, and the sclera, and finally the interior of the eye.

Tuberculosis of the conjunctiva may damage the eye very seriously and not infrequently leads to blindness; it may also form a source of infection for the rest of the body. As it starts as a local disease, if it were possible to heal the primary lesion the prognosis would be better. Treatment consists in killing off the tubercle bacilli. The best means is cauterizing or excising the tissue; the resulting destruction may lead to symblepharon and other deformities. In addition to the local treatment, general treatment is of importance. Tuberculin has been used with advantage by some.

CORNEA.—Aside from the deep keratitis which accompanies scleritis, the cornea is affected in the tuberculous patient in a form of keratitis characterized by circumscribed opacities in various layers of the cornea. These opacities enlarge irregularly, become vascularized and are usually accompanied by very little inflammatory reaction. There will often be associated deposits on the posterior surface of the cornea which will be described more fully later, and are quite typical of a tuberculous process. A small proportion of interstitial keratitis is due to tuberculosis; the patients show other tuberculous manifestations, and syphilis can be excluded. Syphilis and tuberculosis are not infrequently combined in the victims of interstitial keratitis. In this mixed form of interstitial keratitis superficial grayish infiltrates appear, turn yellow, break down, form ulcers which become vascularized and heal. Atypical forms of keratitis are especially suggestive of a tuberculous origin. The opacities are not diffuse but spotted, rather subepithelial. The conjunctival vascularization suggests a superficial keratitis but the epithelium remains intact and deep vessels develop (Heine). A tuberculous irido-cyclitis is often accompanied by deep circumscribed opacities in the cornea; the characteristics of the former condition, and especially the posterior corneal deposits confirm the diagnosis. The disease runs a very chronic course; it is often one-sided and occurs at other times of life than the syphilitic keratitis. The process is sometimes superficial like the extension of a scrofulous pannus and the opacities become yellowish infiltrations.

Sclera.—Tuberculous scleritis is secondary to disease of the anterior uvea. This explains the frequent complicating corneal process known as sclerosing keratitis—which leave permanent tongue-like opacities. The congested scleral area shows a distinct nodular prominence which is usually red and painful and disappears without leaving a trace; its precise nature is not understood, though the application of the galvano-cautery is of advantage. Small transparent vesicles like phlyctenules are often present on the episclera which shoot up as rapidly as they disappear.

Scleritis was formerly regarded as always rheumatic; now the pendulum tends to the other side, and all cases are regarded as tuberculous. There are, however, unquestionable cases of episcleritis which are not tuberculous but are toxic in origin, and yield promptly to a correction of diet. The tuberculous variety show on careful examination manifestations (deep keratitis, changes in the pectinate ligament, nodules in iris) indicative of a deep-seated and severe process. Curiously enough there patients are often fat, well-nourished women who appear in excellent health. The diagnosis is confirmed by the tuberculin test. In the writer's experience this class of cases is unusually amenable to tuberculin treatment. In deep scleritis where the process in the sclera is followed by that characteristic slate-like discoloration and the anterior segment of the eye is generally involved, the scleritis is only a part of a severe intraocular process. In one case which reacted to tuberculin, the writer succeeded in growing the tubercle bacillus from the aqueous on a Miller-Petroff medium.[1]

UVEA.—Tuberculosis of the iris and ciliary body is frequent in young adults or children with a suspicious heredity and suggestive previous history. It is possible to distinguish three clinical forms: (1) perhaps the most characteristic is the form of disseminate nodules. In this, in one-third of the cases, the condition is bilateral. The number of nodules varies from 1 to 20. They may attain a diameter of several millimeters and are usually situated near the iris angle. They are yellow-gray in color, grow slowly and sometimes coalesce, or in some cases the nodules are hidden by a layer of exudate. The entire iris can be transformed into a nodular mass which fills the anterior chamber. The nodules enlarge or retrograde leaving atrophic areas in the iris stroma. It must be remembered that nodules appear in the iris which are not tuberculous. The second variety is characterized by no macroscopic changes in the iris, though there is great tendency to posterior synechiæ. The important symptoms are the posterior corneal deposits. These at first are small and grayish-white, then yellowish. They coalesce to form irregular figures (mutton-fat) or gravitate to the bottom of the anterior chamber like a hypopyon. As they retrograde the color becomes gray, then brown, and finally nearly black, and they gradually become irregular in shape. At the site of large posterior corneal deposits the adjoining cornea may become affected in the form of a deep opacity which remains permanent and occasionally leads to an anterior adhesion of the iris.

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[1] Arch. of Ophth., Vol. XLV, p. 285.

In this group belong the **heterochromia** cases in which change in color of the affected iris is the most striking symptom of the irido-cyclitis. With this change in color the iris tissue undergoes superficial atrophy and the framework becomes outlined like a corrosion specimen. The pigment seam at the pupil is irregularly defective, and the pupillary margin is notched. In Heine's statistics of heterochromias 50 per cent. had cataract, and 10 per cent. glaucoma. This author states that slight heterochromia with partial atrophy of the pupillary margin is the most frequent associated symptom of cataract in adolescents. There are but few adhesions, and on careful examination posterior deposits will be found on the cornea. Trans-illumination may show defects in the pigment layer of the iris and there is usually a complicating cataract. In cases of this character which the writer has observed a history suggestive of tuberculosis was obtainable and the patient reacted to tuberculin. The removal of the cataract was performed with success and a very satisfactory degree of vision obtained in most of the cases. A nearly constant complicating symptom and one pointing to the involvement of the ciliary body, is the associated increased tension. The involvement of the ciliary body explains the serious vitreous changes which occur in these cases.

The third variety is the severest, that of the tuberculous granuloma. This occurs only in children. The anterior chamber becomes filled with grayish-red granulation tissue and perforation occurs at the periphery of the cornea. The cornea is involved in the form of a diffuse opacity, infiltrations, or a typical interstitial keratitis.

With the aid of the strongest illumination and a magnifying glass Stock and Heine have described minute gray nodules occurring at the lesser iris zone and at the pupillary margin. Stock regards them as tubercles even though they may disappear in a few weeks. Heine has described them in a form where there are stroma thickenings near the ciliary border. Finally, there is a form of tuberculous iritis where the iris is not different from that found in the ordinary iritis. With an active tuberculous focus in the iris and ciliary body an optic neuritis may be present and recognized if an ophthalmoscopic examination is possible. This seems to be a toxic process[1] and not an actual tuberculous manifestation indicating the extension of the tuberculous process to the optic nerve and meninges.

Tuberculosis of the uvea usually develops slowly and the condition may exist for months before the patient seeks relief. The symptoms are therefore mild, consisting in a disturbance in vision, tearing, photophobia, moderate congestion and slight or no pain. Usually only one eye is involved. The inflammatory symptoms may be absent or are usually very slight. The iris when there is a single large tubercle, appears practically normal; sometimes it is somewhat discolored, with distinct blood vessels, its markings erased, with a variable number of posterior synechiæ or the anterior lens capsule is covered with an exudate. The tubercles usually appear as such from the beginning.

This disease runs a slow course, the symptoms increase, the nodules enlarge, the process may then become stationary or retrograde; in other cases it progresses and leads to perforation of the eyeball, or makes enucleation necessary. The disappearance of a nodule in the iris is recognized by the absence of the pigment of the iris. The patients are usually children up to the tenth year. The disseminate form is more frequent and occurs between the twentieth and thirtieth year. An examination for possible foci in the rest of the body, according to Groenouw, has shown in 116 cases that 34 per cent. were healthy, 32 per cent. were suspicious, and 34 per cent. were distinctly tuberculous. In a certain number of cases tuberculosis of the iris followed an injury of the eye, usually a slight trauma, such as a blow or a superficial excoriation.

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[1] Trans. Am. Oph. Soc., 1906, p. 215.

The prognosis for vision is bad, and a definite number of eyes must be enucleated. A moderate amount of vision is retained by only a small number. In 131 cases 22 per cent. recovered, according to Groenouw. The vital prognosis is also not particularly favorable. In the statistics just mentioned 18 per cent. of the patients died after a number of months from the general trouble.

Choroid.—There are a number of forms of choroiditis which are in etiological relation to tuberculosis. The one which has been known for the longest time is that occurring in *miliary tuberculosis*. The eyes are externally normal, the media are clear and vision is not affected, as far as it is possible to examine these somnolent patients. Examination of the eye ground shows one or more pale, rounded areas with a pink periphery and a whitish-yellow center. These areas may attain the size of the disc but never show any pigmentation. The vessels of the retina pass over them and the larger foci are distinctly prominent. They are usually situated at the posterior pole, though not infrequently as well in the periphery. A characteristic feature is the rapid appearance and growth of these tubercles. After twelve to fifteen hours new foci can be observed which formerly were invisible. As this occurs particularly in acute miliary tuberculosis, it is usually seen in children, and lesions are visible a few days before death. After their development the patient dies in a few days. Choroidal tubercles may be the first signs of a latent tuberculosis.

The ophthalmoscopic diagnosis is not difficult. Its value in differential diagnosis was perhaps formerly overestimated. It has recently been shown, however, that these changes in the choroid occur much more frequently than was formerly believed if the cases are examined repeatedly and with the help of the electric ophthalmoscope. Marple,[1] confirming a prediction of Stephenson and Carpenter, reported them present in practically all of the cases.

Conglomerate tubercle of the choroid without associated iritis is very unusual. An excellent picture of this condition can be found in Haab's atlas. With the ophthalmoscope a prominent whitish area is seen several disc-diameters in size, with an uneven nodular surface and with small miliary-tubercle-like areas about its periphery. The retina is detached by the tumor and optic neuritis is present. The patient generally dies; in other cases the process perforates the sclera. This picture may resemble a choroidal sarcoma when the tumor is situated in the external layers of the choroid and presents a grayish-blue appearance.

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[1] Ophthalmoscope, 1917, p. 559.

There is another form which can be called **tuberculous granuloma** of the choroid with inflammatory symptoms. Iridocyclitis with nodules in the iris is frequently associated. The inflammatory symptoms may be very pronounced or the case may resemble a glioma of the retina. The tumor invades the sclera and finally perforates the eye ball with the formation of a growth outside of the eye or a subconjunctival abscess. The patients are under twenty, in very poor general health, and usually present other manifestations of tuberculosis.

Exudative Choroiditis.—Though a single patch of choroidal exudate can occur from any septic focus in the body, this form is also seen in the tuberculous—*i.e.*, in those with a suspicious history and a positive tuberculin diagnostic test. It is difficult to say whether the ophthalmoscopic picture is characteristic. There is very little tendency to pigmentation. At the periphery small tubercle-like foci (satellites) are sometimes present. Carpenter and Stephenson have described this variety under the term "obsolescent choroiditis." The fact that a patient with a choroidal exudate reacts to tuberculin does not mean that the choroidal process is tuberculous unless a local reaction takes place. Systematic examination of cases with a patch of choroidal exudate has in my experience shown that they do not react to tuberculin. There is one form of exudative choroiditis, a more advanced form than the above, which in my opinion is definitely tuberculous. In this there are many vitreous opacities and an unusually large choroidal exudate can be faintly recognized. The eye does not respond to treatment, detachment of retina, diminished tension and a cataract develop without much change in the anterior segment of the eye.

Disseminate Choroiditis.—Cases of disseminate choroiditis are probably more frequently tuberculous than is generally supposed. The ophthalmoscopic characteristics are not easily defined. There is perhaps a general tendency for a smaller amount of retinal pigmentation than is usually present in this form of choroiditis, there are fewer vitreous opacities and the blood vessels in the exposed choroidal atrophic areas are not so visible. The foci are indefinite in outline. According to Leber[1] the various forms of disseminate choroiditis can not be told apart. Occasionally the diagnosis is aided by some accompanying signs of anterior uveitis (nodules or atrophies of iris, posterior corneal deposits). In some of these cases satisfactory results have been obtained with tuberculin therapy. A tuberculous etiology is suggested when the case seems otherwise negative.

Finally, mention must be made of cases of **detachment** of the **retina** occurring in eyes which are not myopic and where the detachment is secondary to a choroidal process. This choroidal process may be easily seen or it can not be definitely made out because it is situated too far forward in the fundus. The vitreous is always affected. Though these cases are not cured, decided improvement can be obtained in some by tuberculin treatment. The course and outcome of these cases is different from the usual retinal detachment.

The frequency with which the choroid is involved in general acute miliary tuberculosis makes the immunity of the retina in this disease particularly noticeable, as in septic embolic processes the conditions are reversed. There are only very few reports of miliary tuberculosis of the retina.

Stock, in his investigations on rabbits, found that follow-

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[1] Graefe-Saemisch-Hess, II ed.

ing an intravenous injection of tubercle bacilli, iritis and choroiditis regularly developed, but retinitis was never observed. In chronic tuberculosis of other organs we sometimes find tubercles in the retina, usually as a complication of a tuberculous inflammation of the iris and of the ciliary body. In eyes with normal anterior segments retinal tubercles are very unusual, just as unusual as they are in general tuberculosis.

Retina.—It has been shown in recent years that the retina in young individuals is not infrequently the site of a tuberculous process which is characterized by perivasculitis of the veins. Vision is diminished and the fundus examination shows opacities of the vitreous, and hemorrhages either in the vitreous or in the retina. On careful examination a distinct vascular lesion is found. The walls of a vein for a short stretch will be outlined by a grayish-yellow exudate, which in place looks like an injection of the lymph space. There may be a few surrounding hemorrhages and the blood current seems interrupted. When the process is healed the site of the lesion will be marked by a choroidal atrophy running alongside of the vessel. In these cases tuberculin treatment has been unusually satisfactory.

A somewhat more pronounced variety is found in the so-called **recurring vitreous** and **retinal hemorrhages** in **adolescents.** The tuberculous nature of this condition was developed entirely from other clinical factors, the positive tuberculin reaction, the presence of tuberculous lesions in other parts of the eye and in the rest of the body. Exceptionally, in addition to the circumscribed infiltration of the walls of the veins with its consequences, a number of small round nodules have been observed, situated near these vessels, which did not show progressive development and are regarded by Leber as abortive miliary tubercles of the retinal vessels.[1]

It is very remarkable that these so-called tuberculous changes have never led to involvement of the neighboring retinal tissue. Fleischer (1914), has shown anatomically that in many of these cases the vascular disease of the veins is a tuberculous one, and Leber believes that in the preponderating number of these recurring hemorrhages this is the cause.

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[1] Igersheimer, 1912. Knapp, 1913.

These retinal hemorrhages in youthful individuals are often combined with sudden large vitreous hemorrhages and with a tendency to relapses. Another peculiarity is its prevalence in the male, and the disease usually occurs in men between eighteen and thirty, though some cases have been reported in children. The disease usually begins in one eye and the other is then affected after the first has recovered or while the first still continues to be affected, and so the condition frequently changes from one eye to the other.

The hemorrhages are rarely limited to the retina and vitreous hemorrhages are very much more frequent. These occur suddenly and may within a day or two reduce vision to perception of light. In other cases the change consists in opacities of varying degrees with corresponding diminution in sight. The absorption takes place according to the amount of blood extravasated and may take from a number of months to a year. Improvement is often very noticeable in even the pronounced cases. Relapses are frequent and in some cases have been noted as often as ten or twelve times, so that the disease may continue with alternating improvement and deterioration for many years. I have recently seen a woman of fifty-five who had had recurring attacks of vitreous hemorrhages for thirty years. Finally a certain amount of useful sight may be retained. As a rule, the hemorrhages occur independent of any action on the part of the patient. As they absorb, opacities remain in the vitreous. Sometimes there are white deposits of connective-tissue-like nature which cover the disc and a part of the eye ground, giving the picture of a proliferating retinitis and frequently associated with detachment of the retina and cataract. It is striking that in this disease glaucoma rarely supervenes, differing in this regard from the condition following thrombosis of the central vein.

The disease is a very serious one and the prognosis is often dubious. The hemorrhages have no particular characteristics. They occur frequently in the anterior parts of the fundus and it is impossible to find the vessel from which they seem to be derived. Sometimes hemorrhages have been observed next to a vein and to inclose the vein, so that the origin from the retina seemed probable. Careful examination of the peripheric veins in the retina has shown changes which are probably the cause for the hemorrhages (Eales, Friedenwald). In these cases white sheathes and small circumvascular infiltrations were found with distention of the small veins, with obliteration, tortuousness and the formation of new vessels. The patient, in every other respect, seemed to be healthy, though sometimes they are delicate and anemic. Occasionally a slight cardiac hypertrophy and a tendency to constipation have been present; habitual nose bleed was sometimes a symptom. Hemophilia was suggested as the cause for these hemorrhages, as some of the patients have shown a tendency to bleeding after slight injuries. A retarded blood coagulation has also been observed, though this has not been in any way constant enough to explain the hemorrhages. Others have regarded the hemorrhages as due to hereditary syphilis. The association with tuberculosis was suggested by Axenfeld and Stock in 1909. The tuberculous origin is often supported by the family history and the previous history of the patient, and in some cases nodular iritis was observed. These nodules are sometimes so small that they can not be seen except with a magnifying glass. Tuberculosis was demonstrated by the positive result of the tuberculin injection. Tuberculin in these cases must be cautiously given as the local reaction may result in an increase of the hemorrhage, with resulting serious damage to the delicate retinal elements.

Another variety of tuberculous retinitis is a form of **exudative retinitis** in which the main opthalmoscopic feature is one or more retinal exudates which are apparently in some relation to the veins, as they follow their course, though there are no signs of perivasculitis and hemorrhages are not an important feature. The diagnosis here is made by exclusion, and by the tuberculin test. The exudates are placed in the deeper retinal layers under the retinal vessels and do not produce any change in the choroid. When they are absorbed some sclerosed tissue remains in the retina.

A different and somewhat deeper placed process is the **retinitis** with **massive exudation** (Coats). The etiology of this condition is obscure, and there is no proof that it is tuberculous, though some of the cases have reacted to tuberculin.

In tuberculous irido-cyclitis **optic neuritis** may develop, and can be seen with the ophthalmoscope if the media are clear enough. This need not necessarily mean that the tuberculous process is extending to the optic nerve and that the meninges will in turn be affected, as this seems to be a toxic condition as is seen after inflammation or injury of the anterior segment of the eye.

A **tuberculous papillo-retinitis** with distinct tumor formation at the disc, solitary tubercle, at the nerve-head, is unusual, though a number of cases are on record. The differential diagnosis from glioma is often impossible, as the retina is detached early in the course of the disease.[1] In some cases where the choroid was also involved it is difficult to say whether the process at the nerve-head was a primary condition, though in most cases of tuberculous growth at the disc there are no signs of an origin from the choroid. As a rule the process does not extend into the optic nerve beyond the lamina cribrosa.

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[1] Durst, Inaug. Dissert., Leipzig, 1909.
Knapp, Arch. f. Ophth., Vol. XXXII, p. 22.

Rarely tuberculosis of the choroid and of the retina may give the clinical picture of a purulent panophthalmitis or of a septic irido-cyclitis, particularly when due to a mixed infection. It is furthermore not improbable that a tuberculous perivasculitis might involve the central retinal vein during its course in the optic nerve. In this case the symptoms of a thrombosis of the central vein could be produced together with certain inflammatory signs at the head of the optic nerve.

ORBIT.—According to Michel, in tuberculosis the orbit is affected particularly along the temporal half of the lower margin. The lid is red and swollen, the bone is thickened; fluctuation develops and pus escapes. The probe detects rough bone. In other cases fistulous passages develop and a sequestrum forms. The resulting cicatrization may cause an ectropion of the lower lid. The bone surrounding the orbit particularly the zygoma and the cranial bones may be involved. Tuberculosis of the wall of the orbit usually causes exophthalmus.

Tuberculous involvement of the **lacrymal gland** is very unusual. The clinical picture is not necessarily characteristic. The gland slowly enlarges in the course of a few months to the size of an almond without inflammatory symptoms or pain. There may be some ptosis and swelling of the adjoining glands. As a rule the patient is under thirty. It can be shown that the lacrymal gland no longer functionates, by the demonstration by means of blotting paper of the cessation of the lacrymal secretion (Schirmer).

VI. DISEASES OF THE CIRCULATION

RETINAL VESSELS

Pulsation.—On examining the normal eye ground, a venous pulse will be found present. This is a diastolic presystolic pulse and is the reverse of the arterial pulse. The arterial pulse is a pathological condition when the diastolic pressure in it equals the intraocular pressure, and occurs (Groenouw[1]) as (1) compression pulse, or intermittent entrance of blood in the eye. The arteries at the disc show pulsation when the intraocular pressure is increased by conditions within the eye (glaucoma) by pressure on the outside of the eye ball (by the examiner's finger), or compression of the retinal artery (tumor of orbit and retrobulbar neuritis), or when the heart force is weakened (syncope). (2) True arterial pulse is observed beyond the confines of the disc. The arteries dilate and elongate, increased tortuousness indicating increased vis-a-tergo, in low degree the reflex streak on the arteries broadens rhythmically. This pulsation occurs in aortic insufficiency, aortic aneurysm, Graves' disease, anæmia, etc. Pulsatory movements of the cornea have been observed in keratoconus. Capillary pulse occurs at the disc in aortic insufficiency.

Changes in Calibre.—The arteries are normally slightly smaller than the veins, the relation is ⅔ to ¾ to 1 (Leber). There are certain congenital anomalies which must not be regarded as instances of hyperemia. One of these anomalies consists in **abnormal tortuosity of the retinal vessels.** The convolutions are situated in the same retinal plane, the dilatation of the vessels is uniform and not pronounced in proportion to the tortuosity. In pronounced hyperemia the vessels describe steep arches in the direction of the surface of the retina, whereby they dip into the depths. The steeper these depressions are, the greater foreshortening does the vessel present, and the blood column is broader and darker, so that in the course of the vessel there are alternating light and dark areas.

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[1] Groenouw, Graefe-Saemisch, II ed.

Congenital tortuosity of the retinal vessels, particularly of the veins, is not infrequent. There is no change in the disc. The change is present in both eyes and to the same degree. In the pronounced cases the ophthalmoscopic picture is an unusually striking one. The veins after they leave the disc show a remarkable maze of convolutions and turns, which is in striking contrast to the perfectly straight course of the arteries. The picture is even more striking when the arteries are also involved. The condition is often observed in hypermetropic eyes, especially when combined with astigmatism. Leber says that in 5 cases complicating telangiectasis of the eye region was present and in 2 of these the conjunctiva was involved. The cases of telangiectasis of the retina are probably examples of this condition.

Aside from local disturbances in the retina, there are circulatory disturbances due to general causes. In *anæmia*[1] the retinal vessels may appear translucent with the arteries differing only slightly from the veins and the fundus appears yellow and granular.

Cyanosis of the Retina.—In acquired cardiac lesions which lead to venous congestion in the circulation, hyperemia is usually absent in the retinal vessels. On the other hand, in congenital lesions of the right heart (patent foramen ovale) which lead to general cyanosis, the retinal vessels show a marked distention and a cyanotic color which has given rise to the name of retinal cyanosis.

Another form of general cyanosis is not congenital and in which no cardiac lesion is present, but is due to an abnormal condition of the blood. This is known as *polycythemia* (see p. 413) or erythrocytolysis, in which the number of red corpuscles is very much increased while the leucocytes are about of the usual number. In some of these cases there is an enlargement of the spleen and of the liver, albuminuria and an increase of blood pressure. In the cases of congenital heart lesion which produce cyanosis, investigations have shown the regular presence of a high grade polycythemia. In other words, this condition is common to the two varietes of general cyanosis, though they are to be strictly differentiated.

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[1] Salzmann, Michel's Jahrb., p. 826, 1912.

Ultravenous condition of the blood is particularly apparent in the conjunctiva, where the veins are unusually dark, almost black in color. With the ophthalmoscope the arteries are found to have a color like the veins usually have, while the veins show a very much darker tone. The disc is reddened, with blurred margins. In pronounced cases the vessels are unusually tortuous and run a corkscrew-like course. Vision is usually normal. With pronounced arrest of the circulation, the presence of retinal hemorrhages is easily understood.

In the condition due to polycythemia, of which only a few cases have been observed, the congestion in the eye ground is not so marked and the distention of the veins is not so pronounced. The veins are dilated to double their usual size, with tortuousness, and of abnormal dark color. The arteries are normally wide and present a somewhat darker color.

After circulatory disturbances, particularly occlusion of some part of the venous system in the retina, the veins show circumscribed dilatations particularly in the part peripheric to the obstruction. This represents an attempt to establish a collateral circulation. Leber objects to the term "varicosities" by which this condition has usually been described, and suggests "collateral cirrosities of the veins." The condition has been shown to depend on venous thrombosis. It occurs in persons of advanced years who suffer from arteriosclerosis or cardiac lesions, also in cases of recurring retinal hemorrhages in adolescents. It has been described in syphilis, retrograding choked disc and chronic glaucoma. The condition is usually only in one eye and if seen early hemorrhages and marked venous congestion are present. If the obstruction is near the disc, cirrosities often form a complicated convolute of dilated small vessels. If a retinal branch has been occluded, distinct communication with a neighboring branch can be seen.

Hemorrhages in the Retina.—These occur in changes in the blood or in the vessel walls: the main causes are,[1] in order of importance, chronic nephritis, diabetes, arteriosclerosis, purpura, tuberculosis, scurvy, polycythemia—then anæmia, leukemia, endocarditis, sepsis, and very rarely typhoid fever. Furthermore, in circulatory disturbances of local or general origin: pulmonary stenosis, mitral insufficiency, embolism or thrombosis, hemorrhages in the newborn and during menstrual periods.

It is often difficult from the ophthalmoscopic examination to find a cause for the hemorrhages or to determine from which blood vessel they arise. Changes in the vessel wall can sometimes be seen. The hemorrhages are frequently in close apposition to the vessels, especially the veins. It seems probable that most retinal hemorrhages result from diapedesis and not through rhexis,[2] though they may result from spontaneous rupture of small arterial branches with atheromatous changes, possibly the result of softenings, with the formation of miliary dissecting aneurysms. In the preretinal hemorrhages of the macular region the size of the hemorrhage suggests the origin from a larger vessel, and in a number of cases a vein directly above the hemorrhage has been found affected, though its course was not interrupted and no thrombosis occurred.

Small retinal hemorrhages are rapidly absorbed, the margins become pale and the hemorrhage shrinks. If they are of larger size, they become darker in color, split up and gradually disappear. Somewhat larger hemorrhages may partly change into white or whitish-yellow spots, which change usually begins in the center and gradually enlarges. If the hemorrhage reaches a decided size, especially with recurring attacks, connective-tissue bands and membranes are formed. It is very unusual for the hemorrhages to transform themselves into pigment spots. As a rule pigmentation in the retina is the result of proliferation of the retinal pigment epithelium.

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[1] Heine, Augenuntersuchung bei Allgemeinerkrankungen, II ed., Jena, Fischer, 1910.

[2] Leber, Graefe-Saemisch-Hess, II ed.

The chances of restoration of sight depend on the site of of the hemorrhage and the amount of destruction which the retinal elements have suffered. It is well known that the prognosis in preretinal hemorrhages is very good, as the retina itself is not directly injured. The absorption often requires many months, particularly if a thick layer of fibrin develops at the site of the hemorrhage. In other forms of hemorrhage the prognosis for vision must be guarded, because we can not judge of the degree of tissue damage from the ophthalmoscopic examination. If the hemorrhages are complicated with obliteration of the vessels, absorption will be retarded and atrophy of the retina is frequently the final result. Retinal hemorrhages, in addition to their possible effect on vision, have great importance from their prognostic significance for life, as they occur in conditions which frequently terminate fatally. In this consideration it is well to remember that, briefly speaking, retinal hemorrhages occur in blood changes (anemia, etc.) and in disease of the blood-vessel wall (arteriosclerosis). If blood changes can be excluded, retinal hemorrhages, particularly if associated with raised blood pressure, are an evidence of disease of the blood-vessel wall even if this is not apparent to the ophthalmoscope (see arteriosclerosis, p. 348) and present the same prognostic significance as cerebral apoplexies. The retinal arteries and those supplying the basal ganglia are end arteries and as branches of the internal carotid artery both are exposed to changes in blood pressure in this vessel. At the same time sclerosis in the cerebral vessels is very much more frequent than in the retinal vessels. Uhthoff found retinal hemorrhages in only 4 per cent. of cerebral apoplexies; though if the retinal vessels show endarteritis, a similar condition always exists in the cerebral vessels.

While retinal hemorrhages are very common in retinal arteriosclerosis and an increase in their number occurs with the onset of other serious symptoms, their occurrence has not been explained. Moore[1] believes that the principal factor is an impairment of the nutrition of the vessel walls, and to changes in the blood, and not to increased arterial pressure. (1) He believes that the pressure in the retinal arteries is not increased in arteriosclerosis; (2) a marked increase in the number of hemorrhages is not infrequent toward the end of a patient's life when there is no corresponding increase of blood pressure; (3) while retinal hemorrhages are exceedingly common in leukemia and certain anemias, conditions in which there is no increase of blood pressure.

Geis[2] found the blood pressure raised in many cases of retinal hemorrhages when the hemorrhages were caused by arteriosclerosis, chronic nephritis, diabetes (without plaques), while in lues, anemia and in some cases of diabetic and albumenuric retinitis the blood pressure was normal or even sub-normal.

Geis[3] was able to follow up 68 cases of retinal hemorrhages where they were precursors of cerebral hemorrhages in 50. These were divided into groups of 32 patients with retinal hemorrhages where other symptoms of arteriosclerosis were present and the blood pressure was elevated up to 240. Other causes, like diabetes and lues, could be excluded, and the retinal hemorrhages were regarded as strictly due to arteriosclerosis. The patients' ages varied from between forty-seven to seventy-five. All 32 suffered from apoplexy, and death resulted generally after three to four years from cerebral arteriosclerosis.

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[1] Moore, Retinitis of Arteriosclerosis and Its Relation to Renal Retinitis and to Cerebral Vascular Disease, Quarterly Journal of Medicine, 37 and 38, 1916–17.

[2] Geis, Kl. M. f. A., 1913.

[3] Geis, l. c.

The second group included 8 cases of retinal hemorrhages due to diabetes (not diabetic retinitis). All of these cases were seized with a stroke before the termination of the third year, and death was not later than four years. The blood pressure was always found increased. In the third group, there were 10 cases where no etiological cause could be found; arteriosclerosis, however, was suspected, although there were no definite signs. In these cases apoplexies occurred in all and death ensued after from three and one-half to seven years.

Of the remaining 18 cases, 5 died of cardiac lesions. In 4 cases the retinal hemorrhages were due to lues. The patients varied in age from twenty-three to seventy-four years of age, and all escaped cerebral lesions during an observation period of from five to nine years. In these cases the blood pressure was always normal. Of the nine patients who remained healthy, one had recurring hemorrhages in the vitreous, and in one no other cause than slight myopia could be found; while in the other cases the hemorrhages were either preretinal or occurred isolated in the macula. Isolated hemorrhages in the macula are often due to local processes, and together with preretinal hemorrhages are not to be regarded as possessing the same significance as hemorrhages from the retinal arteries.

Geis concludes that retinal hemorrhages in arteriosclerosis, diabetes and chronic nephritis are usually precursors of brain hemorrhages, which, however, may not occur for a number of years. The prognosis as to life need not therefore be very unfavorable. It is very important in retinal hemorrhages to investigate the blood pressure. Its increase is a serious symptom.

Vitreous hemorrhages have an entirely different significance than retinal hemorrhages, because they occur in a great variety of conditions. If it can be shown that they are due to arteriosclerosis, chronic nephritis or diabetes, they become of serious consequence as far as the likelihood of cerebral hemorrhages is concerned.

Conjunctival hemorrhages are of no importance; compared to hemorrhages from the retinal arteries, they are of less moment than vitreous hemorrhages. Produced by external causes and on bodily exertion they occur at all ages, even in earliest childhood. Recurring hemorrhages in advanced years can not necessarily be regarded as indicating vascular changes and as precursors of cerebral apoplexies.

Hemorrhages From General Venous Stasis.[1]—Conditions which produce general venous stasis, even if sudden and of high degree, rarely produce retinal hemorrhages. After severe injuries of the body, in which the thorax or the abdomen are compressed, it is frequent to find the head, neck and chest and the visible mucous membranes dark blue and covered with small hemorrhages. In a few cases hemorrhages have also been observed in the retina. Roenne[2] has observed hemorrhages occurring in 7 out of 60 cases. This rarity is explained by the protection which the retinal vessels derive from the ocular pressure. As a rule the retinal hemorrhages in these cases cause no visual disturbance and are rapidly absorbed.

Occasionally and more frequently than retinal hemorrhages visual disturbances of cerebral character are observed with a normal ophthalmoscopic picture or a few retinal hemorrhages. The patients, after a period of unconsciousness, sometimes find themselves blind; this blindness is usually transient, lasting from a few minutes to half an hour. In others the restoration of vision is only partial, and optic atrophy, either partial or total, results. The cause for this condition is not understood.

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[1] Leber, Graefe-Saemisch-Hess, Vol. VII, 1, p. 513.

[2] Roenne, Kl. M. f. A., XLVIII, 1910.

Retinal hemorrhages have been described after coughing and vomiting, though this is very uncommon.

Retinal Hemorrhages Through Congestion of the Central Vein Following Massive Intracranial Hemorrhages.[1] — In this unusual condition the blood extends from the subarachnoid space into the optic nerve sheath and distends it. This is then supposed to exert pressure upon the trunk of the central vein. The pressure necessarily must be pronounced and can only occur in large hemorrhages and those with acute onset. They usually are the result of the rupture of a larger artery and as a rule death ensues without an opportunity for ophthalmoscopic examination.

Fleming[2] has shown by numerous autopsies the frequency of this occurrence in fractures of the base of the skull. Uhthoff[3] has described two similar cases and states that the fracture does not involve the bony optic canal. The hemorrhage in both cases was from the middle meningeal artery. Fleming has found that large cerebral hemorrhages which are not traumatic in origin, if they break into the subarachnoid space, are followed by similar changes. The retinal hemorrhages in fractures of the base may appear in the form of the large pre-macular type and this may occur in both eyes and also in non-traumatic cases.

EMBOLISM AND THROMBOSIS OF THE CENTRAL RETINAL ARTERY

Sudden and complete interruption of the central retinal artery causes an instantaneous cessation of the retinal function. The impairment of vision depends in extent on whether the main trunk or one of the branches is occluded. The occlusion is promptly followed by nutritional disturbances and tissue changes, which after a brief time are not capable of recovery; permanent blindness and atrophy of the tissues result.

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[1] Leber, Graefe-Saemisch-Hess, Vol. VII, 1, p. 520.

[2] Fleming, Edinb. Med. Journal, XIII, 1903.

[3] Uhthoff, 29 Versamml. d. Ophth. Ges. Heidelberg, 1901, p. 143.

The circulatory disturbance gives a characteristic ophthalmoscopic picture which can be described as that of an acute ischemia. The cause for this, notwithstanding a similar ophthalmoscopic picture, may vary. It may be produced either by an embolism, spontaneous thrombosis, vascular spasm or obliterating disease of the vessel wall. Furthermore, compression of the artery or division of the nerve may produce this picture. The cause for the disturbance of the circulation can often not be determined during life, though in many cases an embolus has been seen with the ophthalmoscope. Otherwise the cause must be determined from other clinical symptoms.

Effect on Vision.—In some cases the blindness after occlusion is so complete that every sense of light is excluded. Sometimes some vision remains in a small temporally-situated part of the field, and the patient is able to count fingers excentrically. These cases form the transition to those of partial occlusion, where certain vessels are still supplied with blood and consequently some function of the retina is preserved. Not infrequently during the first days or weeks a slight improvement of vision takes place, particularly when it was not completely lost at first. The field enlarges, though as a rule central vision is never regained to any marked degree.

The **ophthalmoscopic picture** is very well known. The arteries are contracted and appear like fine, red lines whose course can only be followed for a short distance from the disc. The slightly clouded wall of the vessel is often outlined by delicate white streaks. The optic disc, on account of lack of blood, is white and dull. Pressure on the eye does not elicit either venous or arterial pulsation. As these pulsations depend on the existence of a circulation, their absence can be of great importance for diagnosis, especially to determine the cessation of the blood current in cases where the vessels are not narrowed. Sometimes the blood column, particularly in the larger veins, splits up into individual segments which are separated by colorless plasma.

Their arrest indicates the cessation of the blood current. Occasionally these segments will be observed to move a short distance, then to stand still, and again to return to their previous position. When the main trunk is occluded, it is rarely possible to observe the embolus directly. This is explained by the fact that the site of predilection for the embolus is the lamina cribrosa.

The ischemia is followed by nutritional disturbance and degeneration of the affected area. This is recognized by a clouding of the retina, which is one of the characteristics of the ophthalmoscopic picture. The cherry-red color of the macula is, as has been long known, a constant symptom. In a short time the amount of blood in the retina increases, particularly in the veins, and pronounced venous hyperemia may be present. This is due to the retardation of the circulation and may exceptionally give rise to numerous retinal hemorrhages. Hemorrhages are otherwise very unusual and generally consist in a few red spots or streaks about the disc. The pupil is dilated and does not react. In the course of a few days the haze of the retina is lost, the vessels enlarge and the circulation is to a certain extent reëstablished. The red color of the macula is lost, and this region assumes a brownish color and there may be yellowish or white spots which finally change to dark, granular pigmentation.

The vessels usually remain permanently narrow, the walls become clouded, the small blood column is sheathed in white lines, or the entire vessel may be transformed into white bands. The disc gradually shows a uniform white color. In some cases a small amount of vision remains in the temporal periphery. There have been cases where vision has been lost suddenly with the ophthalmoscopic picture of an embolus, in which, however, the circulation became rapidly better and the vision improved. In many cases the circulatory disturbance is limited to only one part of the vessel. In these cases the ophthalmoscopic changes coincide with those found in embolism of the trunk, except that the area affected is a circumscribed one. The appearance of numerous hemorrhages in this circumscribed area, suggesting a hemorrhagic infarct, is more likely due to an associated venous thrombosis.

When a branch of the artery is occluded the embolus can be frequently directly observed and appears like a bright round or angular body. In the later stages anastomoses often develop between the occluded arterial branches and the surrounding ones. The characteristic white opacity of the retina and the cherry spot may also be present in branch embolism.

In the embolism of the main trunk with preservation of the macular branches the blindness at the beginning is generally complete. There is rapid improvement which is followed by a more or less complete restoration of vision. In some case the macular fibres are preserved from the beginning. This preserved part of the retina may include the macula or only the area which extends from the optic nerve to the macula. The isolated embolism of the vessels supplying the macula lutea is the direct antithesis to the condition which has just been described. This is an unusual condition and Leber states that 8 cases have thus been reported in which half of them depended upon an embolism of the macular branches of the central artery, and the other half on an embolism of a cilio-retinal artery.

In embolism in old people the opacity of the retina may be absent. The reflex immobility of the pupil and the absence of retinal pulsation on pressure may be the only symptoms.

Arterial thromboses give the same symptoms as embolism. The thrombosis is usually not visible. A similar picture is observed in quinine amaurosis: narrow vessels, clouding of the retina, secondary atrophy with obliterated vessels; and similar conditions can be found after pronounced loss of blood.

Prodromal Attacks.[1]—In many cases attacks of transient blindness precede the final attack. Their frequency can be estimated at about 1 in 4. In most of the cases only one prodromal attack was observed. Those unusual cases in which there are many transient and fleeting attacks which may occur daily or several times during the day are probably not due to embolism but to a vascular spasm. As a rule these attacks affect the same eye. Generally the attack is recovered from though occasionally there is a partial loss of function. These rapidly acting attacks of blindness, Leber believes, are particularly suggestive of embolism for the soft consistency of the occluding mass, permits of a complete retrogression of the disturbance. Cases have been observed where the occlusion consisted in a soft movable mass, presumably atheromatous débris, which was seen to move on further along the vessels and disappear. The cases of transient blindness probably due to spasm of the retinal arteries are characterized by great frequency of attacks and by the long length of time (sometimes for years) during which they appear. These cases are, moreover, frequently associated with migraine and its characteristic scintillating symptoms which are probably due to vasomotor disturbances.

Bilateral Appearance.—Embolism of the retinal artery is nearly always one-sided. Bilateral embolism of the central artery has been rarely observed, usually with an interval of time between the affections of the two eyes, still more rarely simultaneously. A simultaneous blindness of both eyes which was permanent has now been described in 4 cases (Leber), three additional cases of bilateral blindness are reported in which the complete blindness was not a permanent one, and vision in one or both eyes improved. In 24 cases there was a time interval between the involvement of the two eyes. The bilateral appearance is a very unusual one, though it is difficult to obtain any definite information from the statistics. Harms[1] has, however, collected 20 cases where the second eye was lost by a similar process in a variable length of time after the affection of the first eye. Kober[2] reported on 60 cases which occurred during thirty-seven years in the Tübingen Eye Clinic; among these 3 were bilateral.

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[1] Schnabel and Sachs, Arch. f. Augenh'l., Vol. XV, 1885; Archives of Ophth., 1885.

Complications.—Numerous retinal hemorrhages sometimes occur. This suggests a complication with venous thrombosis. In this condition it is usually the rule that the venous thrombosis is not the primary condition but that there is at first an incomplete occlusion of the artery, which causes a slowing of the blood current and possibly through the action of inflammatory agents (Leber) produces a secondary thrombosis of the central vein. Secondary glaucoma has been observed a number of times without a complicating venous thrombosis. Another complication is iritis.

Pathology.—The anatomic examination in many cases showed a pronounced endarteritis and this has been assumed by some investigators (Haab[3] and others) to be the primary disease and the obstructing mass, a secondary condition. This was furthermore confirmed by not finding any occluding mass on histological examination and the closure seemed to be entirely due to endarteritic proliferation.

Reimar[4] believes that the picture of so-called embolism can be produced by a simple proliferation of the intima, and that many features in the clinical picture can only be explained by this assumption. In an examination of all the reports, he doubts the presence of an embolus and does not think a primary endarteritis can be excluded. Harms[5] in a review of these same cases, believes that Reimar has gone too far, while Dufour and Gonin[1] decided conservatively that the field of embolism should be somewhat restricted. Leber's[2] view is that the recent objections to embolism can all be overcome and that embolism should occupy by right as prominent a position as it did at first.

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[1] Harms, Graefe's Arch., Vol. LXXXIV, 1914.

[2] Kober, Deutschmann's Beiträge, No. 85, 1913.

[3] Haab, Deutsche Ophth. Ges., XXVIII Vers., 1900, p. 209.

[4] Reimar, Archives of Ophth., 1900.

[5] Harms, Graefe's Arch., Vol. XLI, 1905.

Leber has endeavored to discover a source for an embolus in all the cases where an embolus was found in the central retinal artery on anatomic examination. He found in the 23 cases a valvular cardiac lesion in 15; arthritis in 1; apoplexy in 2; chronic nephritis in 3; thrombosis of internal carotid in 2; and diabetes in 1, and he concludes that a source for the embolism was present in all of these cases. The clinically observed cases have shown that a disturbance of the cardio-vascular system was present in 70 per cent. of Fischer's cases[3] while Kern, in 1892, found that 66 per cent. of the cases from the Zurich Klinik showed *no* cardiac or any other lesion which could be made responsible for an embolus, but many features pointing to local vascular changes and thrombosis of the retinal artery.

The question of embolism or thrombosis is therefore still unsettled, though most authors are inclined to accept a primary disease of the arterial wall which then leads to closure through obliterating endarteritis or thrombosis. Of particular interest is that in the 30 per cent. remaining cases of sudden one-sided blindness with the picture of embolism no cause can be found. The patients have been in previous good health and later on show no signs of a circulatory disturbance, so that it is not probable that a cardiac lesion or other organic disease has been overlooked. It is particularly striking to note the frequency with which this condition occurs in young people, and especially in females. Leber found in 26 of these cases which he collected that only 4 were over sixty-seven years of age; of the remaining 22, 11 were girls between eighteen and twenty-four-

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[1] Dufour and Gonin, Encyclopédie françâise d'ophth., Vol. VI, p. 739, 1906.

[2] Leber, l. c.

[3] Fischer, Leipzig, 1891.

and 5 were young men between ten and thirty. In some of the women there was anæmia, menstrual disturbance and recurring hemorrhages from the nose. The frequent occurrence in anæmic young women was particularly noted by Gunn and Collins.[1] A peculiarity of this condition is the frequent involvement of a branch rather than the main trunk (Fischer)[2].

The **prognosis** is generally unfavorable. The eye remains blind, though there may be a partial restoration of function which occurs either spontaneously or by treatment. Most results seem to have been obtained from massage. According to Leber, 14 cases have been reported in which complete or nearly complete recovery took place and in 12 where there was decided improvement. Pressure is exerted through the closed lids on the eye and rotating movements are effected. This procedure is to be repeated a number of times daily during the early days, and if any improvement occurs, it would be kept up. The reduction of the intraocular pressure can be obtained by paracentesis or iridectomy. It would be best to start with energetic massage; if this is without result, especially if the signs point to an incompletely interrupted circulation, a paracentesis or iridectomy could be done.

Geis[3] expresses the general prognostic significance of this condition as follows: The picture of sudden occlusion of the central retinal artery can be referred to as sclerotic arterial disease when it occurs in patients over forty years without other etiology and no heart lesion. In these cases this ophthalmoscopic picture has the same prognostic importance which the arteriosclerotic changes of the retinal arteries possess. Apoplectic attacks, however, seem to occur somewhat later. (2) Occlusion of the retinal arteries occurring after forty years in the presence of a heart lesion do not offer a definite prognosis. (3) Occlusion of the central artery or one of its branches occurring before the thirty-ninth year, independent of the cause, have no prognostically unfavorable significance.

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[1] Gunn, T. O. S., 1896. Collins, T O. S., 1897.

[2] Fischer, l. c.

[3] L. c.

THROMBOSIS OF THE CENTRAL VEIN

The ophthalmoscopic picture of this condition has been recognized for many years as a typical one, and is described by Leber as hemorrhagic retinitis. It is caused by a congestion of the blood through thrombosis or an occlusion of the central vein or of its branches, as was first pointed out by von Michel. This thrombosis always occurs in the same place, namely, in the region of the lamina cribrosa.

In addition to the venous thrombosis in a large number of cases an incomplete occlusion or a distinct narrowing of the central retinal artery or of its retinal branches has been found present, which Leber regards as probably the primary condition, the consequent retardation of the blood current being an essential factor. The narrowing of the artery is caused by an endarteritic thickening of the wall, following, according to Leber, a partial embolism in some of the cases; this explains the rapid blindness and the one-sided appearance of the disease.

Complete thrombosis of the central vein causes a very typical clinical picture characterized by rapid though incomplete loss of vision, arrested circulation with marked venous congestion and massive hemorrhages, a tedious and unfavorable course and the subsequent onset of secondary glaucoma. Vision is usually reduced to counting fingers at a few feet; it may improve though as a rule it diminishes and is lost with the onset of the glaucoma. The ophthalmoscopic picture is so well known that it is not necessary to describe it here, except to draw attention to yellowish-white areas which develop a short time after the onset of the trouble in between the large hemorrhages. These sometimes coalesce and form a large white surface; in other cases they may be entirely absent. As a rule, both in size and in number, they are less marked than the hemorrhages, whereby this form of retinal disease differs from that found in albuminuric retinitis. There are various grades of lesser development of this picture, depending upon incomplete thrombosis of the retinal vein. v. Michel has described an incomplete thrombosis and one lesser in degree, marked only by congestion. This author diagnosticates a thrombosis from a dilatation and the deep color of the veins. Leber[1] regards the effect of pressure on the eye upon the retinal vessels of particular importance in the incomplete thrombosis. When this pressure is exerted it will be found that the circulation is not entirely obstructed, though no distinct arterial pulse can be elicited. The contents of the large venous branches on the disc can be emptied, showing that there is not a complete closure of the vein. In some cases where the circulation is partly preserved the blood current can be broken up into segments.

Thrombosis of a branch of the central vein is also a well-marked and easily recognized condition in which the changes are localized to the area of distribution of one of the branches of the vein, though the changes are not entirely restricted to this one branch, as the other vessels of the retina often show slight changes. The ophthalmoscopic changes are those which are found in thrombosis of the trunk, except that they are limited to the area of the vessel involved. The visual disturbance depends upon the extent of the retina involved and whether the macula is included or not. A pronounced defect in the field may indicate an associated lesion in the artery and increase the gravity of the prognosis. If a venous branch has been obstructed for a long time, anastomoses with neighboring branches can occur. This is of great importance as vision may thereby often be preserved.

The course of the condition is usually a chronic one. The hemorrhages absorb slowly and fresh hemorrhages often occur. The optic nerve subsequently shows a characteristic white discoloration. In cases in which there is an incomplete obstruction, the course is more favorable and vision may be regained to a certain extent.

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[1] Leber, l. c., p. 373.

An important complication is an incomplete or only temporary occulsion of the central artery or one of its branches through embolism or endarteritis. This is so frequent, especially in the severe cases, that a close association seems plausible. In the sudden onset of blindness the ophthalmoscopic picture seems to be that of a mixed embolism and thrombosis. The arterial occlusion explains the sudden blindness and the marked arterial ischemia with characteristic retinal clouding; while the thrombosis explains the marked venous stasis and the numerous retinal hemorrhages.

A very frequent complication of the severe cases is secondary glaucoma. This sets in with inflammatory symptoms, miotics are of slight action, operations usually do not avail, and an enucleation is generally the outcome. This secondary glaucoma occurs so frequently after complete occlusion of the central vein that a connection between these conditions must be assumed. The cause of this inflammatory condition Leber believes to be found in the action of injurious substances which are in solution in the ocular fluids and which stimulate an adhesive connective-tissue proliferation with new vessel formation in the angle of the anterior chamber.

The affection of both eyes at the same time or after one another belongs to the greatest rarities. It is, however, not infrequent for one eye to have hemorrhagic retinitis and that after a number of years retinal hemorrhages will be observed in the other eye. These cases usually occur where retinal hemorrhages are due to general disturbances like Bright's disease, diabetes, etc.

Recent examinations, particularly of Coats, Harms and Verhoeff, show the great regularity with which a venous thrombosis occurs always at the same place, namely, at the lamina cribrosa. This condition is frequently associated with a marked narrowing of the central artery or of its branches, which, according to Leber,[1] is an important cause in the development of the thrombosis. The arterial constriction is generally the result of an endarteritis, and must be incomplete in order to produce the typical symptoms of venous thrombosis, as venous hyperemia and hemorrhages demand the continuance of a certain amount of blood circulation. From a mechanical standpoint thrombosis of the retinal vein occurs from slighter pathological changes than thrombosis of the central artery, as coagulation is favored by small obstructions such as bending of the vessels, constriction of the vein, etc. In addition to this retardation of the blood current for which a cause is found in the artery, in many cases an inflammatory origin of the thrombosis is assumed, a local disease of the venous wall. If we assume an inflammatory origin, the fact that in 84 per cent. changes in the artery have been found, the possibility exists that the process regularly begins in the artery and the inflammatory agent is furnished by the arterial lesion. On the other hand, a direct action of the inflammatory agent on the vein is very probable.

In many cases, especially in adolescents, a cause for the thrombosis can not be found. In most cases there are diseases and disturbances of the circulation, particularly arteriosclerosis, more rarely cardiac lesions. This explains their preponderance in later life. Occasionally there is a history of preceding articular rheumatism. The condition has also been observed in Bright's disease, gout, diabetes, leukemia, and after certain infectious diseases. In chlorosis, thrombosis of the central vein has been described similar to marantic cranial sinus thrombosis and thrombosis of the larger body veins.

Excluding the cases with a heart lesion or marasmus, local inflammatory changes must be assumed as present where the disease of the venous wall can not be regarded as a symptom of a general disease which eventually would produce a sclerosis of the cerebral vessels. Cases of venous thrombosis accompanied by sclerotic changes in the retinal arteries, often recognizable with the ophthalmoscope, offer the same unfavorable prognosis that independent disease of the retinal arteries does. A group of cases remain where the vein alone is affected.

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[1] Leber, l. c., p. 414.

Geis investigated the future history of 52 cases of venous thrombosis without distinct sclerotic arterial disease, to decide whether this condition was the beginning of a cerebral arteriosclerosis. In 20 of these between the ages of forty-nine and seventy, attacks of apoplexy occurred, in some before the thrombosis, usually between the first, second and fifth years, and in some after nine, ten or twelve years. This author finds that in 50 per cent. thrombosis of the retinal vein is a local disease, as in half of his cases, after an observation period of five years or more, no symptoms of cerebral sclerosis occurred. In 40 to 50 per cent. the venous thrombosis was a forerunner of a cerebral sclerosis, which in some of the cases did not show itself before the ninth, tenth and twelfth year.

It is evident that a thrombosis readily occurs from slight changes following general diseases and in the constituency of the blood, especially if the blood current is retarded through some local physiological change. Even if symptoms of general arteriosclerosis accompany a thrombosis of the retinal vein, the venous thrombosis is not always a precursor of sclerosis of the cerebral vessels, nor is the presence of albumen in the urine necessarily of evil prognosis. In cardiac lesions and in advanced years a thrombosis of the retinal vein is of no prognostic importance. Venous thrombosis usually remains localized to one eye, and very exceptionally does the other eye become affected. In Geis' 52 patients venous thrombosis occurred in the other eye in only 4.

ARTERIOSCLEROSIS

The retinal vessels are the only cerebral vessels which are visible and the only vessels where we are able to recognize the slightest changes. Owing to their character as end arteries, they resemble the blood vessels which supply the large ganglia whose lesions lead to apoplexies and softenings; they are equally exposed to the effects of blood pressure in the internal carotid artery and to the injurious agents which lead to diseases of the vessel walls.

In general arteriosclerosis, changes in the retinal vessels of moderate degree are frequently observed. Raehlmann,[1] who has made extensive investigations on this subject, found in 210 cases in nearly one-half, changes in the retinal vessels (white sheaths, circumscribed contraction or dilatation). This condition has been confirmed by Friedenwald, Lurje and Hertel. The latter two examined anatomically the retinal vessels in patients with marked general arteriosclerosis, who had, however, had no symptoms pointing to the eyes. The changes which Hertel[2] found in 14 cases were partly senile and partly arteriosclerotic. The former occur as well in individuals of a corresponding age who are not suffering from arteriosclerosis and were constantly present and in all parts of the vessel, the arteriosclerotic changes were characterized by appearing in circumscribed areas, and of varying degree. Their presence was sometimes only suggested; in other cases it was very pronounced, though usually in only a definite location and not throughout the entire extent of the vessel. In the trunk of the central vessels these changes consisted in thickening of the intima with increase of the elastic elements, particularly of the artery, though sometimes these changes were of a more inflammatory character. In only a few cases were the changes pronounced enough to produce decided narrowing of the lumen of the vessel. On the whole the changes in the branches of the retinal vessels were slight.

Lurje[3] in a similar series of 18 persons with pronounced arteriosclerosis, particularly of the cerebral arteries, found

[1] Raehlmann, Zeitsch. f. Augenh'l., Vol. VII, p. 425, 1902.
[2] Hertel, Deutsche Ophth. Gesellsch., XXVII, Vers. 1900.
[3] Lurje, Inaug. Dissert. Dorpat, 1893.

changes in the retinal vessels. These consisted in thickening of the intima by a new formed connective-tissue layer between the endothelium and the elastica interna, not only in the trunk of the artery, but particularly in the retinal branches, though in only one case was the lumen distinctly narrowed.

Changes in the retinal vessels occur in those cases of general arteriosclerosis in which the large cerebral arteries are distinctly affected. Gunn found in 24 cases of hemiplegia the retinal arteries on ophthalmoscopic examination normal in only 4.

While in cases where arteriosclerosis in the retinal arteries has been found on microscopic examination, a pronounced sclerosis of the cerebral vessels, frequently with softening of the brain, has been found present; in other cases with pronounced sclerosis of the cerebral vessels, these changes had gradually diminished in the direction of the periphery and were not present in the very delicate terminal vessels, namely those of the retina. This shows that though the eye ground may be normal to the ophthalmoscope, it does not necessarily follow that the cerebral arteries are normal. Furthermore, the changes in the retinal vessels may be so slight that they can not be recognized with the ophthalmoscope. Hertel says that a positive finding of sclerosis of the retinal vessels assumes a similar condition in the cerebral vessels with certainty, but not the reverse. In a number of cases where pronounced vascular degeneration was present in the cerebral vessels, the ophthalmoscopic condition was normal, and atheromatous changes of the large cerebral vessels do not necessarily lead to changes in the retinal vessels. It seems as if there were a direct dependence of disease of the vessels of the eyes on the parent vessels at the base of the brain. Some authors have described the first ophthalmoscopic changes of arteriosclerosis of the retinal vessels to be slight tortuousness, slight changes in calibre, and a more distinct vessel reflex, and have regarded these changes as of serious prognostic importance for the condition of the cerebral vessels. Uhthoff has shown that this view is too extreme, that these changes may occur in the normal and be physiologic. Furthermore, there are slight changes in the retinal arteries (increase of elastic fibres) which are to be regarded as a purely senile change. Raehlmann and Gunn have drawn attention particularly to variations in calibre of the blood column.

On Ophthalmoscopic Evidence of General Arterial Disease.—Gunn[1] says that ophthalmoscopic changes in an advanced case show arterial changes, signs of mechanical pressure on veins, retinal œdema and hemorrhages. The arteries are abnormally narrow with irregularity (dilatation) of their breadth; their course is unusually tortuous. The central light streak is narrow and very bright; this is important in the case of the secondary and tertiary branches of the central artery; their entire breadth seems light in color. When the artery crosses a vein, it loses translucency and exerts manifest pressure on the underlying vein. This is shown by the vein losing its central light streak on either side, or by its being slightly pushed to one side. Later the venous current is impeded with peripheric engorgement of the vein. Œdema of the retina shows itself by want of the full red fundus reflex; a decided gray haze may be present.

Deyl thinks that hypertension can be demonstrated by noting the increased degree of pressure required to produce arterial pulsation. Moore[2] states that in some cases of retinal arteriosclerosis a surprisingly small amount of digital pressure is required to elicit pulsation of the retinal artery. In these cases this author believes the pressure in the ocular arteries is very much below the normal, though the general blood pressure may be twice its normal height, and that this reduced pressure with a disease of the endothelium leads to thrombosis of the central retinal

[1] Gunn, T. O. S., 98.

[2] Moore, R. L. O. H., 1915.

artery. He concludes that in some cases of general arteriosclerosis with high general blood pressure the blood is ultimately distributed to the tissues at a pressure which is actually less than normal.

The early stages, according to Thoma, show increased tortuosity of the arteries, distinct presence of true arterial pulse and a progressive peripheric venous pulse. Gunn[1] has drawn attention to a peculiar glistening appearance which the arteries present in arteriosclerosis, suggestive of sliver wires. The light reflex is unusually narrow and bright, not only near the disc but in the branches of the second and third order, which gives the impression of unusual fullness and rounding. Gunn believes that a narrowing of the reflex streak and the greater rounding of the artery is indicative of increase of vascular pressure. The silver-wire arteries are regularly present where there is an increase in the general arterial pressure. Gunn furthermore has pointed out the impression which the sclerosed artery exerts on an underlying vein. The vein apparently underneath this position contains no blood and there is a peripheric dilatation. A similar observation was made by Raehlmann, who believes that this pressure causes a circumscribed sclerosis of the venous wall. Leber[2] suggests that the vein becomes invisible because it is depressed down into the tissue underneath the artery, and that it can not be definitely accepted that pressure on the vein produces a distinct congestion. Moore[3] in discussing the characteristic changes of the retinal vessels which Marcus Gunn[4] first pointed out as being indicative of retinal arteriosclerosis, emphasizes the fact that the changes are of greatest importance when they involve the tertiary and secondary branches of the retinal artery. The symptom of obstruction of the blood flow in the veins when they are crossed by arteries is in Moore's opinion the most valuable and important single sign of severe arteriosclerosis.

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[1] Gunn, T. O. S., 1892.

[2] Leber, l. c.

[3] Moore, Retinitis of Arteriosclerosis and Its Relation to Renal Retinitis and to Cerebral Vascular Disease, Quarterly Journal of Medicine, 37 and 38, 1916–17.

[4] Gunn, T. O. S., Vol. XVIII, p. 356.

Gunn observed the evidences of obstruction to the flow in the veins when they are crossed by sclerosed arteries, but did not insist upon the importance of displacement of the line of the vein at such crossing. Moore finds that at the arterio-venous crossings there are not only (*a*) evidences of obstruction to the flow in the veins, but (*b*) displacement of the line of the vein. When the vein is twice crossed by the same artery within a short distance the trapped portion of the vein is apt to be very much reduced in size; and while in the retina the veins usually cross deeper to the arteries, occasionally when a vein is observed to cross over a highly sclerosed artery its actual narrowing can then be seen. In the early stage of sclerosis of the artery the line of the vein will remain unaltered, the vein being hidden under the artery. As the arterial wall becomes thickened, the line of the vein becomes diverted, and the displacement is proportionate to the degree of sclerosis. In marked arteriosclerosis when a vein and artery converge to cross obliquely, the vein as it approaches the artery will be found to change its direct course and become diverted, so as to run alongside of the artery; then after it has passed the artery, it will continue to run alongside of the artery before resuming the line of its former course. In the case of very severe sclerosis of the vein where the vein meets the artery at a very acute angle, it will be noted that the vein will pass underneath the artery at right angles to the latter's course. The same phenomenon Moore finds is present even when a vein crosses superficially to the artery. In leukemia where a distention of the veins is frequently present, a lateral displacement has not been observed.

de Schweinitz[1] speaks of the following signs as being suggestive of retinal arteriosclerosis, "and which must not be disregarded if they are associated with other symptoms of arterial disease. Three signs may be seen early: (*a*) A markedly corkscrew appearance of certain arterial twigs, either of those which skirt the macula, or, more significantly, of one or more small branches which arise from the larger vessels of the main distribution which themselves are apparently normal.

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[1] de Schweinitz, International Clinics, Vol. I, 17 series, 1907.

(*b*) "A flattening of a vein where it is in contact with an artery, and at this spot it has somewhat the appearance that a strap would have if laid across a solid tube, provided the vein passes over the artery. If the artery passes over the vein, the appearance is analogous to that which would be produced if the strap were placed across the lower surface of the tube. The vein is only slightly compressed at this stage, but is not yet indented sufficiently to produce an ampulliform dilatation of the vein beyond the point of crossing.

(*c*) "The nerve-head has an appearance often loosely described as 'congested.' "

The diagnosis of congestion of the nerve-head should be made with great caution, as there are such wide physiological variations of congestion compatible with a healthy optic nerve. The latter ophthalmoscopic signs are changes in the vessel wall by which they become thickened, opaque, and are gradually transformed into white lines, which sheath the narrow or obliterated blood current, the lumen becomes narrowed and occluded. Similar changes occur in the veins, though the veins are more tortuous than the arteries in the later stages. Other changes are hemorrhages, œdema, and exudates.

Uhthoff[1] believes that these so-called first ophthalmoscopic signs of retinal arteriosclerosis, consisting of slight tortuousness, slight variation in caliber, and a somewhat accentuated reflex streak, are to be interpreted with caution in considering their prognostic bearing on the state of the cerebral vessels, as these changes can occur in the healthy and be physiological. For the certain diagnosis of arteriosclerosis of the retinal arteries the vessel walls must be outlined with white lines (vascular sheathing) and distinctly thickened, the lumen must be narrowed up to the point of ischemia and complete obliteration; in the earlier stages the diagnosis can only be made when arteriosclerotic changes have led to arterial occlusion through thrombosis, as in the picture of closing of the central artery.

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[1] Quoted from Geis, Kl. M. f. A., 1913.

This represents the most conservative standpoint in the interpretation of the retinal arteriovascular changes. On the other hand, the writer agrees with de Schweinitz in regarding the early signs as suggestive and of importance if associated with other symptoms of arteriosclerosis.

Bardsley[1] was struck by the observation that the changes in the retinal vessels indicating arteriosclerosis sometimes do not have the grave significance which arterial degeneration usually signifies. He observed that these changes sometimes come on very rapidly and in some cases a very remarkable improvement was noted by the disappearance of many of these signs. He therefore believes that the bad prognosis should not be universally applied and that there are conditions which present these changes in the vessels which are not necessarily due to arterial degeneration. After reducing the blood pressure Bardsley was surprised to find that the ophthalmoscopic signs of retinal sclerosis were appreciably diminished. To reduce this high blood pressure the toxic cause, among which he mentions septic stumps in the mouth, should be eliminated.

It is important to distinguish between the vascular changes which are due to simple increased blood pressure and those that are due so vascular fibrosis. According to Bardsley, simple high pressure presents the following picture: (1) the vessels are uniformly distended and full; (2) the light streak is broadened; (3) the light streak is brighter and in conditions of very high pressure it appears like a bright copper wire, in distinction to a silver wire; (4) the tight arteries indent the veins. The signs indicative of sclerosis are the following: (1) irregular tortuosity, particularly of the small twigs; (2) increased brilliancy of the light streak; the light streak appears narrower and more central; (3) irregularity of caliber; (4) general diminution in size of vessels. This author was able by increasing the blood pressure in patients with normal arteries after the administration of adrenalin to increase the blood pressure up to 40 *mm*, and was able to observe the development of the signs just described. In drawing deductions from ophthalmoscopic examinations it is important not to overlook errors of refraction, as they naturally blur the ophthalmoscopic picture, conditions of acute toxemia occurring in acute Bright's or in severe influenza, and finally the existence of the failing heart.

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[1] Bardsley, British Journal of Ophthalmology, April, 1917.

Autopsy reports and clinical observations have shown that very frequently in cases with arteriosclerotic changes of the retinal arteries, softening of the brain has occurred. Geis[1] has followed the subsequent history of 17 patients for several years, to decide the question of how often cerebral complications occur under these conditions and whether the ophthalmoscopic picture allows a definite prognostication of a subsequent cerebral lesion. The ophthalmoscopic diagnosis of arteriosclerosis was absolutely certain, and the changes were present in both eyes. Five of these cases were between forty and forty-seven years of age; 7, between fifty and sixty; 4, between sixty and sixty-seven; and only one was over seventy. All of these 17 patients suffered from apoplexy, generally after the recognition of the retinal lesion, though in some cases the apoplexy preceded or was simultaneous. Death usually resulted from apoplexy, always within four years. This shows that in all of these cases with ophthalmoscopic arteriosclerosis a severe cerebral sclerosis was present, which led to an apoplexy.

Distinct sclerotic changes of the retina (sheathing, thickening of the wall, narrowing of the lumen, obliteration associated with hemorrhages, and exudates, usually bilateral) are a part of the picture of disease of the cerebral vessels which frequently lead to cerebral softening or hemorrhages. The ophthalmoscopic evidence of sclerosis of the retinal arteries has therefore serious prognostic importance.

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[1] Geis, Kl. M. f. A., 1913.

Arterial degeneration of the **choroidal vessels** is not infrequent. The depigmentation of the retinal pigment allows a view of the choroidal arteries. Their walls become thickened, the blood lumen narrowed and finally abolished. Areas of this process are not infrequent about the optic disc, and sometimes a symmetrical area embraces the disc and the macula. The writer has had under observation for five years a case of sclerosis of the choroidal vessels in a man aged seventy, which has remained stationary. There are symmetrical areas of pigment atrophy including the disc and macular region in which a few sclerosed choroidal vessels still remain; the retinal vessels are perfectly normal. Vision is $^6/_{12}$. Th field is practically normal except that green perception is lost.

As the choroidal vessels are entirely different from the retinal vessels, inasmuch as they are not end arteries, their diseases do not permit any deduction on the condition of the cerebral vessels. In four of Geis' cases with sclerosis of the choroidal vessels which were followed for from four to six years, no signs of a disease of the cerebral arteries could be found.

Michel[1] says that arteriosclerosis is a relatively frequent cause of **iritis** in middle and old age which is sometimes associated with the symptoms of a diffuse arteriosclerosis or of a contracted kidney. The iritis presents moderate inflammatory symptoms and signs of disturbed circulation in the iris tissue with dilatation and tortuosity of the larger veins, particularly in the ciliary portion, and with hemorrhages in the iris tissue; sometimes the inflammatory signs are more marked and combined with many small hemorrhages.

This same author makes atheromatous changes in the carotid artery responsible for the development of **senile cataract,** which is often combined with signs of contracted kidney and myocarditis. This view has not been substantiated and is not shared by other authorities.

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[1] Michel, Kl. Leitfaden d. Augenhlk., p. 215.

Arteriosclerosis affects the **choroid** in two forms. In the first it is limited to the macular region in the form of superficial choroidal atrophies and pigmentation, and is usually called senile. When the larger vessels are involved, an area surrounding the optic nerve and macula is affected.

In central senile chorio-retinitis Nettleship[1] distinguishes four well-marked clinical types:

1. Central guttate choroiditis, which is the clinical equivalent of the so-called colloid nodules of the elastic lamina, called Tay's choroiditis.[2] A belt of the spots encircles the disc and macula region, leaving the latter usually free, and vision is not affected.

2. An area of epithelial atrophy occupies the yellow spot. The border is often well defined, the general shape is round or oval. There may be some pigment.

3. Both epithelium and choriocapillaris are destroyed, the large vessels are laid bare, usually altered by opaque thickening of their walls and contraction of lumen; the blood column is obliterated or hidden. The diseased area is sharply defined, circular or oval in shape, and may extend to the disc. In the typical cases there is no collection of pigment.[3]

4. This area is occupied by an oval or round patch of a densely white-gray substance lying under the retina and choroid.[4] The retinal vessels sometimes dip into this mass and disappear.

Nettleship believes that central senile chorio-retinal disease results from disease of the posterior ciliary arteries.

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[1] Nettleship, On the Distribution of the Choroidal Arteries as a Factor in the Localization of Certain Forms of Choroiditis and Retinitis, R. L. O. H., XV, 1903.

[2] Pictured in T. O. S., Vol. IV, pl. 2.

[3] T. O. S., Vol. IV, pl. 8.

[4] T. O. S., XIX, pl. 9.

PRESENTED
BY THE EDITOR OF
M. J.

In retinitis pigmentosa and in some cases of syphilitic retinitis the distribution of the disease is dependent on the arterial system of the choroid at the equator, and the ring-shaped scotoma is probably due to the supply at the equator being the poorest.

Arteriosclerotic Retinitis.—Moore[1] believes that when retinal exudates become engrafted on retinal arteriosclerosis a distinct ophthalmoscopic picture results to which he gives the name of "arteriosclerotic retinitis." It differs from renal retinitis in a number of ways. The most important is the absence of the exudates which are characteristic for renal retinitis. In arteriosclerotic retinitis retinal hemorrhages are almost invariably present. The retinal exudates take the form of small white dots, spots or small areas. They are circular in outline, sharply defined, without any change in the surrounding neighborhood. The dots are sometimes arranged about the radicals of the smallest veins and a favorite site is between the macula and the optic disc. They sometimes form a constellation about the macula, somewhat resembling a partial star figure. The dots are very slow to develop and change slowly. Moore has observed that they may completely disappear without leaving any trace. In addition to the small dots, larger areas or small plaques are observed in the advanced cases, usually in the central region. They resemble the small dots in being a dirty white color and irregularly round. They change but very slowly and sometimes develop cholesterine crystals. In some cases Moore has observed a condition of the arteries which he calls "pipe-stem sheathing." This consists in white plaque-like deposit in the perivascular sheathing which often surrounds the vessel for a part of its course, like a pipe stem. The breadth of the blood stream is quite unaltered, thus differing from the usual conversion of the arteries into white threads with a very narrow blood stream, as seen in arterial disease. This condition was described by Hulke.[1] The changes which Moore describes are present in only one eye, which is an important characteristic. While the dots and areas of exudate seen in this form of retinitis resemble those found in renal and diabetic retinitis, retinitis circinata, their scantiness, their course and their association with severe arteriosclerosis justify in Moore's opinion the suggestion of a new clinical picture.

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[1] Moore, l. c.

As arteriosclerosis frequently affects the blood vessels of the brain, a variety of lesions of the optic pathways results with central disturbances of vision. These same circulatory changes may affect the nuclei of the ocular nerves and thereby cause ocular muscle paralysis.

The **optic nerve** can be mechanically injured through pressure of the altered arterial wall in sclerosis of the internal carotid or of the ophthalmic arteries in those places where these structures are contiguous. This pressure has sometimes been so marked that the nerve has practically been divided in half. There is an optic atrophy which occurs in old people which is apparently of arteriosclerotic origin and is probably explained in a part of these cases by these changes just described.

Moore[2] believes that arterial disease of the retina may cause **optic atrophy.** In these cases the sclerosis of the arteries has attained such a degree that the supply is slowly reduced to a point no longer sufficient for the needs of the retina. While no sudden interference with the retinal supply results, there is a deterioration in sight and a constriction of the field. In these cases the disease of the retinal arteries is always well-marked. There is no cherry spot at the macula, at the early stages some œdema of the disc is present which is later followed by optic atrophy. The œdema is always mild in degree. The constriction which the visual fields undergo are irregular in type. The atrophy has the characteristics of a secondary atrophy, the disc having an opaque white appearance with the edge lacking in sharp definition.

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[1] Hulke, R. L. O. H., 1866, quoted from Moore.

[2] Moore, l. c.

Some authors speak of **arteriosclerotic atrophy** occurring in old people. The disc is pale, sharply outlined, with peripapillary atrophies of the pigment epithelium. The examination of the vessels of the disc shows some sclerotic changes. This condition may also be due to a sclerosis of the ophthalmic artery or of the internal carotid which exerts a pressure on the optic nerve. The VI nerve in the cavernous sinus is then frequently involved. The disturbance of vision is slight, by which this condition differs from the optic atrophy following the closure of the blood vessel.

Glaucoma.—As glaucoma has been brought into relationship with arteriosclerosis, it may be well to consider this connection at this point, excluding the cases of glaucoma due to disease of the retinal vessels, the so-called hemorrhagic glaucoma (thrombosis of the central retinal vein).

It is desirable first to briefly explain that normal intraocular pressure is produced by the blood pressure, which, in turn, is the source of the fluid within the eye. This fluid results not so much from a secretory process but from a transudation of a peculiar kind in which, under normal conditions, certain constituents of the blood serum are retained, as a difference in the amount of albumen contained in blood serum and in the aqueous humor will show. The production of the ocular fluids is to a great extent dependent upon blood pressure, as is the ocular pressure. Wessely has examined experimentally the dependence of the intraocular pressure on the blood pressure. He found that the pulsation of the ocular pressure presented a distinct curve a moment later than that of the carotid. If the blood pressure is increased, the ocular pressure is also increased, though to a lesser degree on account of certain regulations, such as the reduction of blood pressure in smaller vessels, increased lumen of the vessels, elasticity of the eye ball capsule and degree of distention of the eye ball. Contrariwise, the reduction of blood pressure causes a depression of the ocular curve. From this dependence of ocular pressure on general arterial pressure it seems natural to infer that glaucoma could be readily explained by pathologically raised blood pressure and that there must be a close connection between glaucoma and disease of the cardio-vascular and renal systems in which high blood pressure is so important a symptom.

Arterial Tension in Glaucoma Patients.—The writer examined 50 cases of primary glaucoma of which careful records had been kept, and the blood pressure was found to average about 150; it was 140 in patients under sixty years old and 159 in those over sixty. Kümmell[1] examined 70 cases of glaucoma and found the blood pressure in those below sixty was 163.2; over sixty, 169.3; together 165.7. The normal pressure as found in 90 cases without glaucoma and of about the same age showed an average of 145.2; in persons over sixty the average was 152.85, and in those under sixty it was 138.6. This shows that in five-sixths of the cases there was an increase of blood pressure. This author also found a difference in arterial pressure according to the variety of glaucoma. Fourteen cases of acute glaucoma showed an average pressure of 177. This is not unexpected, as a disturbance in the vascular system is present. The pressure varied between 160 and 245. The conditions were similar in the subacute cases; in 18 the average was 175.2. It has been pointed out particularly by the French authors, that blood pressure is increased in the inflammatory forms. The arterial pressure in chronic glaucoma was found increased only to a slight degree (160.6) and lower than in the inflammatory forms, though these cases are usually in old people. In 30 cases the average pressure was 153.3; in 7 of these the pressure was below the average 145 mm. and 4 presented other symptoms of diseases of the cardio-vascular system or of the kidneys.

[1] Kümmell, Graefe's Arch. Vol. LXXIX, No. 2.

Krâmer,[1] on the other hand, found no direct connection between blood pressure and glaucoma and in his investigations there was no difference in blood pressure between the acute and chronic forms of glaucoma. Sattler[2] also could find no regular increase in the blood pressure of his glaucoma patients. He adds that persons suffering from high blood pressure are not specially prone to glaucoma and usually show other eye lesions, particularly often associated with local disease of the vessel walls. He has seen no glaucomatous attacks attributable to increased blood pressure from muscular exercise. On the other hand, the exhaustion and heart failure which follow over-exertion, mental depression and weakening disease such as influenza, are frequently associated with glaucomatous attacks. Löhlein[3] does not believe that disturbances of the cardio-vascular system can be regarded as an essential factor in glaucoma. In many pronounced glaucomas we do not find high blood pressure or venous congestion of head and eye, and the absence of glaucoma in conditions with high blood pressure (nephritis) and blood congestion to the head show that there must be local predisposing causes.

Ocular Tension in General Arteriosclerosis.—The examination of the ocular tension in general arteriosclerosis has given valuable information on the question of this relation. Thus Moore[4] examined 38 patients, the subjects of general arteriosclerosis in whom the eyes showed no abnormality other than sclerosis of the retinal arteries; and found that such variations in the ocular tension as occur are not associated with a like variation in general blood pressure. He compared the intraocular tension of patients with a general blood pressure of 220 or more and those with a blood pressure of 160 and below, and found an insignificant difference. Krämer[5] also examined 14 patients with chronic vascular and renal lesions who presented a very high blood pressure but in whom the ocular tension was normal.

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[1] Krämer, Graefe's Arch. Vol. LXXIII, p. 349.

[2] Sattler, Berl. klin. Wochenschr., 1913, Nos. 49 and 50.

[3] Löhlein, Zentralblatt f. ges. Ophth., 1914.

[4] Moore, R. L. O. H., XX, 1915.

[5] L. c.

Priestley Smith,[1] with characteristic acumen has drawn attention to the important fact that chronic excess of pressure in the radial or carotid arteries is no evidence of excess of pressure in the capillaries that secrete the intraocular fluid; on the contrary, it is usually associated with increased resistance in the arterioles and insufficient capillary circulation.

Starling and Henderson found that a considerable rise in the general blood pressure did not with any constancy produce a permanent rise in the intraocular tension, and Moore concludes that in some cases of general arteriosclerosis with a high general blood pressure, the pressure of the ocular vessels is below the normal. Intraocular tension is presumably dependent on the condition of the ciliary arteries for which the ophthalmoscopically visible retinal vessels are no guide, as a considerable independence is manifested in the degree in which the various systems of vessels in the eye are involved.

Ophthalmoscopic Changes in Glaucoma.—The writer examined the retinal vessels for sclerotic changes in the above-mentioned series of 50 patients, to determine the value of ophthalmoscopic findings in glaucoma. In the 40 cases of chronic glaucoma, arteriosclerotic changes in the retinal vessels were present in 8, and these were all mild in character. In the 10 cases of acute and subacute forms (congestive type) arteriosclerotic changes were present in 8; in the other 2 a satisfactory ophthalmoscopic examination could not be made. It is very striking to note the frequent and very marked changes found in the acute glaucomas after the attacks were past; some of the arteries were nearly obliterated and sheathed with broad white lines and the discs were pale.

Vascular Changes in the Eye.—The question of local vascular changes in the eye with glaucoma is an important one. General vascular disease surely indicates changes in the more delicate vessels in the eye. The results of pathological examination of ocular vessel changes in glaucoma are varying. Bartels[1] acknowledges the frequency of these changes after a critical examination, though he does not think that these changes are sufficient to be specific and are not greater than the sclerotic changes which are to be expected. Arteriosclerosis diminishes in a distal direction; thus the changes must be more pronounced in the orbit than they are in the eye. In hemorrhagic glaucoma Kümmel demonstrated marked changes in the ciliary arteries back of the eye.

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[1] Priestley Smith, Ophthalmic Review, 1911.

If glaucoma be divided into two groups[2] circulatory and nervous, the circulatory group would be characterized by congestive attacks, by retinal vascular changes, by local changes in the eye which favor increased tension, such as small eyeball, age of patient, etc. The treatment of this group is operative and attention to the general health (arteriosclerosis) is of greatest importance.

Intermittent Closing of Retinal Arteries.—Temporary obscurations are not infrequent clinical manifestations of a disturbed retinal circulation, and they often precede permanent, partial or complete blindness.

These attacks of temporary obscuration were formerly attributed to embolism; the plug supposedly at first obstructed the main trunk of the retinal artery, and later lodged in a branch (Graefe, Mauthner, Schnabel and Sachs). This theory was later abandoned,[3] because of the number of recurring attacks without any evidence of the subsequent fate of the embolus (Loring, Wagenmann).

According to Ole Bull,[4] Zehender was the first to draw attention to vessel cramp as a cause for sudden amblyopia and amaurosis. Benson[5] has reported on a case of intermittent obscurations in which he examined the patient during an attack and found the inferior temporal artery to be bloodless. This condition promptly disappeared, and later the ophthalmoscopic examination gave almost negative results. Dr. Benson thought that spasm probably determined the attacks. Wagenmann[1] examined a patient with recurring obscurations during an attack and found both arteries and veins contracted. Iridectomy checked the process for seven months; then an attack with permanent blindness supervened. This author believes that the attacks can only be explained by vascular spasm, the local cause being arteriosclerosis. Galezowski has directed attention to the positive existence of vascular spasm of the retinal arteries and its relation to arterial thrombosis. Ole Bull believes that angioneurosis may be as frequent a cause for circulatory disturbances in the retinal vessels as embolism or thrombosis, and that all cases of obscurations are due to cramp, especially as spasm is likely to occur when pathologic vessel changes are present and often leads to a thrombus formation. Osler[2] states that in certain stages of arteriosclerosis arteries are very prone to spasm.

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[1] Bartels, Zeitzschrift f. Augenh, lk., Vol XIV.

[2] Knapp, Archives of Ophthalmology., 1916.

[3] Leber, Graefe-Saemisch-Hess, II ed., still upholds this theory for certain cases.

[4] Krankh. d. Retinalgefässe, Leipsic, 1903.

[5] Trans. Eighth Internat. Ophth. Cong., Edinburgh, 1894.

Russell[3] in a paper on "Intermittent Closing of Cerebral Arteries," suggests the term closing to express this vessel action and describes loss of brain power from intermittent closing of cerebral arteries (temporary paralysis) as it takes part in the production of permanent paralysis. This closing is caused by vessel changes effected by means of the muscular coat; it is partial or complete, producing impairment or complete suspension of function.

As examples of local closing there is the local syncope of Raynaud's disease. Raynaud[4] states that he observed a cramp of the retinal vessels in the sickness which bears his name, but Panas,[5] at Raynaud's request, examined some of

[1] Arch. f. Ophth. (Graefe), 1897, XLIV, 220.

[2] Allbutt's System, Edition 2, VI, 627.

[3] Brit. Med. Jour., Oct. 16, 1909.

[4] Quoted from Dufour, Encyclop. Française d'Ophthal, VI, 117.

[5] Traité des maladies des yeux, I, 619.

his patients and failed to find any difference in the calibre of the vessels during the access of asphyxia. The contraction of retinal vessels in this condition has, however, been found by a number of other observers.

In this group of vasomotor phenomena belong contractions of the retinal artery in epilepsy, malaria, whooping cough and migraine.

Intermittent closing of healthy retinal arteries may occur without any lasting effect on vision. In diseased retinal vessels this phenomenon is of grave significance, as the vessel may remain obstructed or lead to thrombosis. In subjects with retinal arteriosclerosis, especially if obscurations have been present, a rigid general treatment of the arteriosclerosis should be given, mainly with a view of preventing any of the causes which seem to contribute to closing of the arteries.

Retinitis Circinata.[1]—Goldzieher, in 1887, described a condition under the name of "Hutchinson's Changes of the Eye Ground," which subsequently proved to be identical with the disease which Fuchs was the first to fully describe and which he called retinitis circinata. Hutchinson's disease is really different from retinitis circinata. It is a disease which was described in 1876 by Hutchinson and Tay as symmetrical central chorio-retinal changes in senile persons, in which the pale dots are caused by colloid excrescenses of the vitreous lamina. Subsequently this disease has usually been spoken of by English authors as Tay's choroiditis or choroiditis guttata.

In the clinical picture, Fuchs makes a sharp distinction between the affection at the retinal center, the so-called macular spot, and the crown-like zone of white degenerative foci, the so-called spotted girdle, which two conditions, though associated, do not seem to stand in the same relationship to the cause of the disease.

As a rule there is no connection between this disease and other general diseases; the development of these changes are not understood. de Wecker suggested a hemorrhagic origin, and believes that the white areas result from fatty degeneration, which an anatomically examined case published by Ammann in 1897 confirmed. Leber says that the development of the degenerative foci from hemorrhages can be excluded with great probability, and that these two changes are probably, like in nephritic retinitis, dependent upon a uniform though at present entirely unknown cause. In many cases no hyperemia and, in fact, no vascular changes are observed. The eyes seem normal; there is a gradual diminution of sight; the ophthalmoscopic picture shows a striking spotted circle which forms a horizontal ellipse consisting of small white areas about the fovea contralis. The spots are milky or yellowish-white, glistening and sharply defined. They may be discrete or coalesce to form large white areas. The white areas sometimes show distinct prominence; the retinal vessels pass undisturbed over them. The extent and shape of this circle presents a number of variations. Sometimes we find in addition to the perimacular circle or instead of it, one or more excentrically placed rings.

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[1] Leber, Graefe-Saemisch-Hess, Vol. VII, 2, p. 1242.

The center of the ring in the typical cases is occupied by the so-called macular spot, whose appearance may vary. In the recent cases it is small and sharply limited, and appears as a small white spot with a dark margin. Generally it is a number of disc-diameters in size. The retina in this region is cloudy and grayish-yellow in color. There may be a number of hemorrhages, also dark gray or brown spots. The retinal vessels in this region are not hidden. The tissue change must therefore be deeply situated. Between the macular spot and the girdle there is a zone of red eye ground. In many cases the changes in the region of the macula are not pronounced. At the same time a central scotoma with a marked visual disturbance is always present. In very severe cases the clouded macula presents a distinct prominence which suggests a possible detachment of the retina in this region. Nuel has drawn particular attention to this central detachment. In many cases it is, of course, difficult to decide whether the prominence is due to a thickened retina or to a detachment.

The rest of the eye ground usually shows no particular changes. The disc is usually normal, though it may be slightly congested and not quite defined. The retinal vessels in most cases are practically normal. Though changes in the vessels have been observed by some authors, they do not seem to be capable of explaining the changes in the retina.

An occasional complication are hemorrhages, which may be transformed into whitish foci quite different from the white spots in the girdle. In the periphery of the eye ground colloid excrescenses are sometimes present. These are scattered, yellowish-white, rounded spots, occasionally surrounded by a delicate pigment ring. The vitreous is usually clear. In addition to the hemorrhages, there may be slight dust-like opacities and cataract formation, not unexpected at the age of the patient.

Vision is very slowly involved and the patient can not definitely state the onset of the trouble. It is striking that the patients often do not present themselves before a number of years have elapsed. The disturbance of sight is usually marked and the central vision is completely gone. The field shows no peripheric changes, the light sense is not involved. The course is an extremely chronic one. Occasionally the process will remain unchanged for years or there may be a slow progressive change. According to Leber, some observers have reported on a retrogression of these white areas, which have slowly broken up into individual spots and finally disappeared entirely, giving place to cholesterine crystals. This retrogression in one case was so complete that after four years nothing remained except the colloid excrescences. With the retrogression of these changes no improvement, however, of the vision takes place.

In other cases the process leads to a marked thickening of the retina. Changes are apt to develop which suggest retinitis exudativa in which the subretinal space is filled with a large quantity of fatty degenerated tissue and desquamated pigment epithelium.

Retinitis circinata belongs to the unusual diseases. Among 70,000 cases Fuchs has observed it in only 11. It occurs especially in people of advanced years; two-thirds of them are usually between sixty and seventy years; the average age is 57.3. The disease is one-sided or bilateral. The one-sided cases are somewhat more frequent, the ratio being 4 :3. The condition may be more developed in one eye than in the other and the second eye may not be involved for some time after the first, and there are one-sided cases in which the other eye has shown only retinal hemorrhages, hemorrhagic retinitis or other retinal changes.

The cause is unknown, though its frequent presence in advanced years and the absence of other causes makes it likely to be due to arteriosclerosis. There is no reason to associate the condition either with tuberculosis or with syphilis. It has been observed in pronounced diabetes and in albuminuria. Arteriosclerotic changes of the larger vessels were present in most cases. The diagnosis is easily made, as the picture is characteristic in the pronounced cases, and it can be easily distinguished from nephritic and diabetic retinitis, from colloid excrescences and from the so-called retinitis punctata albescens. Prognosis is unfavorable; the disease tends to advance and is not amenable to treatment. The progress is, however, very slow and usually only central vision is lost.

Retinitis with Miliary Aneurysms[1]

Under this heading Leber describes a disease of the retina which consists in extensive infiltration of the retina combined with multiple miliary aneurysms. Notwithstanding its rarity, quite a number of clinical observations have been published and one case has been anatomically examined by Coats and Morton, 1908. Leber, in 1912, described these cases more carefully and emphasized the differences between this condition and the usual miliary aneurysms which are found in the retina, and showed that the condition usually occurs in adolescents and generally in males.

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[1] Leber, Graefe-Saemisch-Hess, Vol. VII, 1, p. 28.

Ophthalmoscopically the picture resembles retinitis circinata, though aside from the presence of miliary aneurysms, it differs from this condition by a greater irregularity in the distribution throughout the retina, often with a marked development of the changes which may lead to detachment of the retina The condition, therefore, approaches the type of retinitis which Coats has described as retinitis with massive exudation. Coats has described a subdivision of this disease, which presents pronounced vascular changes, particularly in the veins. As miliary aneurysms are only exceptionally present, the changes in the retina are probably independent of them, though there may be a common cause. Eleven cases of this disease have been observed. In many the miliary aneurysms were not recognized and only hemorrhages and a peculiar retinal change were noted. In some cases latent tuberculosis seemed to be present, though for the majority of these cases there is no reason to assume a tuberculous origin, particularly as the ophthalmoscopic changes do not correspond to those usually regarded as tuberculous and in the anatomic investigation of one eye no tuberculosis was found present.

The ophthalmoscopic picture shows a normal disc and the larger retinal vessels are unaffected. The white retinal infiltrations vary in size and in distribution. Occasionally, just as in retinitis circinata, there is a small pigmented area in the macula which is surrounded by a ring-shaped zone of infiltration. In other cases the macula is also included in the area of infiltration or the area of infiltration may extend from the disc in some other direction.

The miliary aneurysms may affect not only the arteries, but also the veins. They are always limited to the part where the tissue changes are present. In the advanced cases detachment of the retina regularly occurs. The vision is very much reduced and the field restricted. Complete blindness usually followed the addition of secondary glaucoma. All cases have occurred in males, between the ages of thirteen and twenty-six. The cause for these aneurysms is not understood.

Pulsating Exophthalmos

This condition is produced by rupture of the internal carotid artery in the cavernous sinus and causes a typical picture characterized by exophthalmos. The upper lid is red, œdematous and swollen; dilated and dark veins are unusually prominent. The lid is tense and hangs down over the protruding eyeball. The conjunctiva is chemosed. The degree of the exophthalmos varies. The eye is usually pushed straight forward with some interference with its motility, particularly to the outer side. On the sclera the veins are unusually prominent. The ocular fundus presents a similar state of venous engorgement, and occasionally a choked disc. Pulsating of the eyeball may be distinctly felt on palpation, and auscultation over the upper half of that side of the head reveals a bruit of which the patient is himself conscious. The two symptoms cease on compressing the common carotid artery of the same side. There are often associated ocular paralyses. The condition may be bilateral, though it is usually one-sided. The cause is an arterio-venous aneurysm within the cavernous sinus which is usually traumatic. A severe blow on the forehead causes a spicule of bone to enter the cavernous sinus and injure the internal carotid artery.

The lesion has also followed shot injuries and severe concussion of the head. Sometimes it is spontaneous from degeneration of the carotid wall. Slight exertion, such as bending over or coughing, may produce a rupture. The pulsating exophthalmos which is observed in women during pregnancy is explained in this manner. The symptoms then develop rapidly after the patient has experienced a snap and pain.

In cases of fracture of the base the general symptoms of this injury at first predominate, then after a number of weeks or even months the pulsating exophthalmos slowly develops. Optic atrophy is the frequent outcome. Spontaneous recovery has been observed and probably consists in a thrombosis of the superior orbital veins. In most cases the condition has required operation.

Intermittent Exophthalmos

This is a condition in which the eye becomes proptosed intermittently whenever a venous stasis occurs in the orbit. This can be caused by bending the head over, by compression of the jugular vein or by forcible expiration. The eye protrudes when the patient bends the head forward, and on assuming the upright position the eye seems somewhat sunken. The trouble is usually one-sided and causes no particular distress. It has generally been believed that this condition is due to a varicose distention of the orbital veins which are affected through a disease of the venous wall.

Birsch-Hirschfeld has suggested the cause to lie in a stenosis of the anterior venous channels of the orbit which normally would permit the blood to escape from the orbit when the head is bent forward, and when the escape of the blood to the cavernous sinus is made more difficult.

The cause for this condition is sometimes given as a severe bodily exertion, whooping cough, or an injury. In many cases no cause has been found. Nowithstanding this exophthalmus, the eye is not affected and changes in the eye grounds are usually absent. The prognosis is usually an unfavorable one, as far as recovery is concerned, though the condition is not necessarily a serious one. Complications have been observed in the form of orbital hemorrhages and visual disturbances which have led to atrophy of the optic nerve.

VII. DISEASES OF THE RESPIRATORY TRACT

HERPES

Herpes of the **Cornea** was first described by Horner. It is a condition usually observed in adults in severe catarrhal diseases of the respiratory tract, in pneumonia, and in any febrile disease; though often it appears independently and without known cause. Simultaneous eruptions of herpetic vesicles may be present on the nose and lips. The eruption appears usually directly after the acme of fever. At first there are a number of isolated clear vesicles on the cornea, though when the patient comes for examination the vesicles have burst and epithelial defects remain. These defects sometimes show a characteristic branching tree-like distribution which is known as **dendritic keratitis.** The cornea in these affected areas is generally anæsthetic, and a decrease in the intraocular tension and miosis have sometimes been noted. Cabannes[1] divides corneal herpes into three varieties: (1) febrile herpes; (2) neuralgic herpes; (3) ophthalmic zona. Neuralgic herpes is very rare. After violent pain for twenty-four hours an eruption of blisters appears when the pain ceases. The blisters heal, the lids are at first red and œdematous, and there are marked signs of conjunctival irritation. The cornea at this stage is hyperæsthetic. Then small superficial infiltrations appear on the suface of the cornea which are ruptured vesicles.

In the **zoster** form of herpes the lids and the forehead are affected in a similar manner and are anæsthetic. These two forms, however, though they differ in degree, are probably related. The condition is usually one-sided, painful and extremely tedious; recovery taking place in from four to six weeks. Relapses are not frequent. The corneal areas denuded of epithelium are liable to secondary infection with all its sequelæ, and the eye often suffers from severe intraocular complications independent of the corneal changes. Schmidt-Rimpler described the first form as febrile herpes, as opposed to the neuralgic herpes which includes herpes zoster and all other afebrile forms of herpes. The lesion in herpes zoster has been placed in the Gasserian ganglion. Gebb has obtained satisfactory results from the use of salvarsan in herpes zoster. In febrile herpes the condition is probably a toxic neuritis of the corneal sensory nerves.

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[1] Cabannes, Soc. franc. d'ophth., 1908.

Superficial punctate keratitis was first described by Fuchs.[1] It is characterized by the appearance of a number of superficial corneal defects and infiltrations which always accompany a severe catarrhal affection of the upper respiratory passages. The condition is usually observed in young adults. The symptoms are those of corneal irritation and are quite severe, though if the infiltrations do not become infected, leading to corneal ulceration, the condition heals promptly without doing serious damage to the cornea. This condition is also placed in the group of neuropathic disturbances of the cornea, and Verhoeff believes that the lesion is located in the ciliary ganglion. In two cases Fuchs found this condition associated with facial herpes. A form of this keratitis is observed in children as a variety of phlyctenular disease where the surface of the cornea seems strewn with sand.

Pneumonia

Pneumonia, like other acute infectious diseases, causes metastatic ophthalmia, optic neuritis and ocular muscle paralysis. These lesions are unusual and do not show characteristic features.

Catarrhal Affections of the Nose and Throat

These are of great importance for the ophthalmologist. It is remarkable how many cases of conjunctivitis and of superficial corneal processes, particularly the marginal forms, are apparently caused and kept up by a catarrhal rhinitis, as is shown by their prompt cure upon treatment of the nose. A frequent condition is an eczema of the nasal entrance which occurs in phlyctenular disease. This complication is varyingly interpreted as an independent condition or as an extension of the conjunctival process along the naso-lacrymal canal. The treatment of the eczema of the introitus nasi is an important factor in the management of these phlyctenular cases. The rhinitis in these cases is frequently kept up by adenoids and they in turn are associated with hypertrophied tonsils. Even in older patients recurring corneal ulcerations may be due to a catarrhal rhinitis kept up by adenoids. The treatment of the eye condition consists in the removal of the adenoids.

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[1] Fuchs, Wiener kl. Wochenschr., No. 44, 1899.

In investigating an association between the diseases of the eye and the nose, Hoffman found in 137 children with scrofulous eye disease, an associated affection of the nose present in 38 per cent. In more than half of these adenoid vegetations were present, in one-fourth there was chronic rhinitis. In fact, in these cases the frequency of nasal affections is striking. Seventy-five of the 137 were operated upon in the nose. The result of this was first of all that the local treatment of the eye was very much more successful than before, and the favorable results of the combined treatment were permanent, as most of the patients on reëxamination had continued to be relieved of their eye condition. The combined treatment of the nose and the eye in these cases is, therefore, of great importance.

Pressure and congestion in the nose may produce a variety of reflex neuroses of the eye. Treatment of the nose frequently causes a relief of eye pain or of œdema of the lids and of the conjunctiva or of persistent conjunctival hyperemia. de Schweinitz has found episcleral congestion to depend upon sinus disease in some cases.

DISEASES OF THE NASAL ACCESSORY CAVITIES

The ocular complications of nasal sinus disease can be best described by regions.

Orbit.—The most striking and probably the best-known orbital complication of nasal sinus disease is that due to a dilatation of the frontal and ethmoidal sinuses without inflammatory signs, the so-called **mucocele,** which causes a mechanical displacement of the orbital contents. The eye ball is generally uninvolved. The distention which the sinus undergoes is associated with rarefaction of the bone, usually of the inferior wall of the frontal sinus. On the other hand, in a case which the writer operated upon, the upper or cerebral wall of the frontal sinus was absent and the altered mucous membrane of the dilated sinus was directly adjacent to the dura of the anterior cranial fossa.

Orbital Periostitis.—Inflammation of the sinuses is associated with external or orbital inflammatory signs whenever the inflammation invades the intervening wall. In the mild cases there is no macroscopic change in the bone, but a periostitis is present over the floor of the frontal sinus with swelling and redness of the eyelid or over the ethmoidal os planum, causing exophthalmos. This condition may resolve of itself or on intranasal treatment. If the involvement of the bone is more active a discoloration or a circumscribed defect in the bone will occur usually in a typical site in the floor of the frontal sinus or at the ethmoidal os planum. The pus thus gains its way to the periosteum and forms a subperiosteal orbital abscess which either remains encapsulated or may extend forward and perforate the skin of the eyelid; the suppuration may then cease, the wound heal, or a purulent fistula remain. The orbital contents are generally not involved in these cases beyond slight venous congestion at the optic disc, as the periosteum is easily detached from the bone and protecting granulations spring up. Subperiosteal orbital abscesses are not infrequent in children, the infection being transmitted by the ethmoid labyrinth.

If the orbital periosteum itself is invaded, which is most likely in the places where it is normally adherent, as for the passage of vessels, involvement of the orbital structures results in the form of cellulitis or abscess. The optic nerve becomes inflamed and the ophthalmoscopic changes at the nerve-head may be more pronounced on the nasal side. This was especially noticed in one of the writer's cases in which the lesion in the orbital periosteum corresponded exactly to an area of necrosis just below the posterior ethmoidal foramen. The orbital condition recovered after the ethmoidal focus was removed.

The important and preponderating part played by nasal sinusitis in producing orbital cellulitis and abscess has, strange to say, been recognized only within the last years.

Birch-Hirschfeld[1] found in 684 cases of orbital inflammation the sinuses involved in 60 per cent., of which the frontal was affected in 28.8, the antrum 21.8, the ethmoidal 20.5, and the sphenoidal 6.1. In 14.7 per cent. several sinuses were affected. The extension of the inflammation usually takes place where the periosteum is thin, where the blood vessels pass through the bone and where dehiscences occur, though the extension may be by means of a phlebitis. Some authors claim that nearly all orbital inflammations are caused by nasal sinus disease. Under these circumstances the usually practised blind incisions into the orbital cavity are not surgical; the treatment should be based on the lines of the orbital operation for nasal sinus empyema, aiming at broad exposure by an incision along the orbital margin, careful inspection of the orbital walls, and proper drainage according to the individual case.

The importance of the maxillary antrum in causing orbital complications has in the writer's mind been exaggerated. Disease of the antrum itself rarely seems to cause an orbital complication unless through the intermediary of the ethmoidal cells. The extension follows much more frequently a periostitis of one of the walls, as after an alveolar abscess. Thrombophlebitis of the orbital vessels, according to Uhthoff, is unusual after accessory sinus disease, and is more frequent from extension of a process in the face.

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[1] Birch-Hirschfeld, Klin. M. f. Augenhlk., 1908.

Optic Nerve.—A very important group of ocular complications of nasal sinus disease are those of the optic nerve. That the optic nerve sometimes suffers in sphenoidal sinusitis has been known for some time, and brilliant cases of restoration of sight have been recorded by Holmes and others by timely treatment of the sphenoidal sinus.

Francis and Gibson[1] examined 100 skulls for the study of anatomic relations between the sphenoid sinus and the orbit. In 38 per cent. the orbit was separated from the corresponding sphenoid sinus by a relatively thin wall, sometimes only ¼ *mm* thick. There is no symmetry between the two sides and the sinus of the one side may extend over so as to come into contact with the optic nerve of the other side.

The recent investigations of Onodi have shown that the optic nerve is frequently in close relation to the posterior ethmoidal cells and that the extreme thinness of the intervening wall makes the involvement more likely than in the case of the sphenoid. While the intervening wall between the optic canal and the sphenoidal sinus varies from 1 to 12 *mm* in thickness, the dividing wall between this canal and the posterior ethmoidal cell may be as thin as tissue paper or measure 1 to 2 *mm.* Cases have been observed that bear out the frequency of this involvement. Of special interest is the involvement of the optic nerve in the form known as retrobulbar neuritis in which a central scotoma is the characteristic functional defect.

No anatomic examination of a case in question had been published until Birch-Hirschfeld's[2] recent article. In this case a carcinoma of the posterior ethmoid cells had perforated into the orbit at the apex and caused exophthalmos and optic neuritis with central scotoma. Histological examination found venous stasis due to compression of the orbital veins by the tumor and isolated disease of the papillo-macular bundle posterior to the site of entrance of the vessels into the optic nerve. This consisted in an œdema of the optic nerve, swelling and proliferation of the glia cells, and disintegration of the nerve fibres. Its cause and localization depended upon venous congestion and toxic action on the nerve fibres, the latter resulting from inflammatory changes present in the tumor. This author believes that the visual disturbance (central scotoma) in disease of the posterior nasal accessory cavities has a similar origin. The venous stasis is explained by an extension of the inflammation to the blood vessels at the apex of the orbit and a direct toxic action on the optic nerve is probable.

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[1] Francis and Gibson, Ophthalmoscope, Vol. IX.

[2] Birch-Hirschfeld, Sehnervenerkrankungen bei Erkrankung der hinteren Nebenhöhlen der Nase, Archiv f. Ophth., Vol. LXV, No. 3, p. 440, 1907.

The optic nerve shows in the beginning no ophthalmoscopic changes unless the nasal infection is situated in the anterior ethmoidal cells, as in a recent case of well-marked papillitis with central scotoma which occurred in the writer's practice. Elschnig[1] found that the ophthalmoscopic changes in optic nerve involvement secondary to sinus disease varies from a perfectly normal fundus to pronounced choked disc.

Van der Hoeve[2] believes that the first symptom of disease of the posterior nasal accessory sinuses is an enlargement of the blind spot. This enlargement is larger for colors than it is for white. This symptom apparently occurs when the posterior ethmoidal and sphenoidal sinuses are involved, as they are so frequently, and is absent when the anterior sinuses and the antrum only are involved. de Kleyn confirms these observations and he found the blind spot enlarged in 47 out of 52 cases of posterior sinus disease. Enlargement of the blind spot is, of course, found in all conditions which enlarge the disc or cause surrounding atrophy or in medullated nerve fibres, myopic conus, choked disc, etc. This symptom, therefore, to be of value, can only be positive when these conditions are excluded. Van der Hoeve believes that the first suggestion is an enlargement of the blind spot for colors, which increases in two directions, either through the development of an absolute peripapillary scotoma for white or the scotoma for colors becoming larger, or the development of a relative central scotoma for colors which may spread and join the blind spot, thus forming the typical oval scotoma. The enlargement of the blind spot and the central scotoma may occur as independent occurrences, though in advanced cases they fuse. This author believes that the enlargement of the blind spot is caused anatomically by involvement of the nerve fibres which take their origin in the retina about the disc. In other words, these are the fibres which occupy the periphery of the optic nerve and on account of this location they are the ones most exposed to inflammatory conditions in the optic nerve sheath. Markbreiter[1] examined the disturbances of the field of vision in nasal sinus disease and found that out of 100 cases in 52 the blind spot was enlarged and in 28 this was the only change. He was not able to confirm the opinion that this symptom differentiates between suppuration in the anterior and posterior sinuses. In the writer's experience the examination for the blind spot has been of distinct value, although the blind spot has not been found enlarged in all cases in which the posterior sinuses were affected; nor should it necessarily be, as this symptom probably depends upon certain anatomic relations of the optic nerve to the posterior ethmoidal cells, which are not always present. The writer has found a convenient way of determining the blind spot by using the Haitz stereoscopic apparatus[1] and by having the patient fix the extreme margin of the card, the outline of the blind spot can then be easily marked out.

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[1] Elschnig, Med. Klinik, Vol. X.

[2] Van der Hoeve, Archives of Ophth., 1911.

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[1] Markbreiter, Z. f. A., Vol. XXXI.

Onodi[2] has drawn attention to contralateral visual disturbances in nasal sinus disease. He has found a number of specimens in which a sphenoid or a posterior ethmoid sinus of one side has been in close contact with the optic nerve of the opposite side, with an intervening wall which was extremely thin. These anatomic peculiarities must be borne in mind in explaining symptoms which occur on the opposite side to that of the nose affected.

Optic neuritis[3] of nasal origin occurs in two forms; one in which the ocular affection develops during the course of a nasal suppuration and the other in which there is a typical retrobulbar neuritis and the rhinoscopic examination is negative. In the latter cases exploratory operation reveals a latent infection. The more acute the case the more rapid is the loss of vision and the better is the prognosis. The favorable effect of the nasal operation is remarkable. The nasal diagnosis in these acute cases is often difficult; even with negative rhinoscopic findings an exploratory operation must frequently be undertaken.

Ocular muscle paralyses are not infrequently the only manifestations of orbital complications. In a case of grippe with ethmoidal involvement which came under the writer's observation the internal rectus for a time was paretic. Posey[4] believes that muscular anomalies are sometimes due to sinus disease owing to the close position of the nerves and the ocular muscles to the walls of the sinus. A number of cases of abducent paralysis in diseases of the nasal sinuses have been described. Thus, Ewing and Sluden[5] produced paralysis of the VI nerve after making an injection into the region of the sphenopalatine ganglion. This paralysis has been observed in a number of cases of sphenoidal disease. Onodi explains this involvement by an unusual course of the VI nerve.

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[1] Made by Sydow, Albrechtstr., Berlin.

[2] Onodi, Z. f. A., Vol. XXXI.

[3] Paunz, Arch. f. Augenh'lk., Vol. LXXV.

[4] Posey, Oph. Record, Vol. XXII.

[5] Ewing and Sluden, Am. Jour. Ophth., Vol. XXXI.

Neuralgia, especially ciliary and retrobulbar, is frequently due to sinus affections, and asthenopia is a common symptom. According to Kuhnt,[1] muscular asthenopia, accommodative asthenopia, and contraction of the visual field, are the functional disturbances observed in nasal empyema in the order of their frequency; he believes them to be due to absorption of toxins from the purulent focus in the sinus. There is a group of eye affections comprising irido-choroiditis, glaucoma, and detachment of the retina, with which certain authors have brought sinus disease in etiological relation. Some over-enthusiastic observers have even cured these conditions by surprisingly insignificant operations on the nasal sinuses. This is a subject upon which we require additional information. Kuhnt, whose experience is surely great, states in the above-quoted article that he has never seen a case of iritis or choroiditis caused by sinus disease, but thinks that cyclitis, especially the form chiefly characterized by opacities of the vitreous, can depend upon this cause. The writer has paid especial attention to this possible connection, but has never seen any definite result from an operation on a nasal sinus on the course of an ocular inflammation except in optic neuritis.

That the list of ocular disturbances which can be produced by sinus affections is not exhausted, there can be no doubt, and a great deal of careful work needs still to be done to complete our knowledge of the relation of nasal sinus disease to disease of the eye.

The Dependence of Nasolacrymal Disease on the Nose

There is still a great deal of mystery about the etiology of lacrymal disease. It is questionable whether the accepted cause, namely, a stricture of the lacrymal duct, is always the cause for lacrymal disease. It must not be overlooked that a stricture can not occur without a sub-epithelial ulceration which probably is secondary to diseases of the periosteum or of the bone. In infants it is not at all infrequent to find a stoppage of the tears on one side. The sac in these cases becomes ectatic and upon pressing over the region of the sac a fluid which varies from glairy mucus to muco-pus is expressed. It has been shown that the cause for this lacrymal obstruction in infants is due to an imcomplete opening of the lower part of the lacrymal duct into the inferior meatus. The condition is not severe because in most cases simple cleanliness and regular expression of the contents of the sac effect a cure. In other cases if this is not sufficient, dilating the punctum and syringing through the lacrymal passage is sufficient to restore the function of the duct. The next age at which we find lacrymal disease in young children is from eight to fifteen years; these patients suffer from inherited syphilis. The disease of the superior maxilla causes a characteristic facial change and an obliteration of the lacrymal ducts. The purulent dacryocystitis in these cases requires surgical treatment and the condition is often a complicated one.

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[1] Kuhnt D. m. W. 1908, No. 37

The lacrymal diseases which are seen later in life are frequently the result in mild cases of a congestion of the venous plexus about the lower extremity of the lacrymal duct, associated with rhinitis. Treatment of the nose and careful syringing of the lacrymal passages with weak astringent solutions are sufficient to effect a cure. The cases in which chronic dacryocystitis and particularly acute lacrymal abscesses develop are, in the writer's opinion, always secondary to an ethmoiditis. Treatment of the nose, if seen early, will relieve the condition. If the condition, however, has existed for a length of time, causing permanent obstruction to the passage of tears, nasal treatment does not avail. In the removal of the lacrymal sac we have been struck by the frequent disease of the periosteum and of the underlying bone found present in the lacrymal fossa; and in the advanced cases there is a communication between the lacrymal fossa and the ethmoid cells.

Tonsils.—As the tonsils become affected in a number of infections, it is perfectly logical to expect toxic inflammations of the eye to follow, such as optic neuritis and paralysis of the ocular muscles. In a case of the writer's an acute retrobulbar neuritis in both eyes developed after a tonsilitis. Mention should be made of the possibility of the infection extending along the intimate venous anastomoses of the pterygoid plexus and causing a thrombophlebitis of the orbital veins and of the cavernous sinus with the symptoms of an orbital abscess and blindness or of exophthalmos and death. Abscesses of the orbit have also been reported. The paralysis of accommodation which has been observed probably depended upon mild forms of diphtheria.

The tonsils give rise to chronic septic infections just as the teeth do, as has been described. The infection then depends on whether the abscess is shut off or not. The diagnosis of an offending tonsil is often difficult, as it may show no manifest signs of infection, the foci of pus are hidden or the tonsil is of the submerged variety and not until after its removal is its diseased condition noted. As a cause for eye disease the teeth are, however, a much more important factor, and the infection of the teeth and of the tonsils is closely related.

VIII. DISEASES OF THE DIGESTIVE TRACT

THE TEETH

In alveolar periostitis the swelling of the cheek is sometimes so intense as to include the eyelids, with closure of of the eye and conjunctivitis. There is usually a point of tenderness along the anterior surface of the superior maxilla which confirms the diagnosis.

Periostitis of the superior maxilla as an extension of an infectious process from the teeth which may involve the orbit, causing the symptoms of an **orbital abscess** in which the lids are swollen and red, the conjunctiva chemosed, the eyeball protrudes and its motility is restricted. As a rule there is no change in the interior of the eye, though sometimes varying degrees of neuritis have been observed and vision is affected. The process usually leads to optic atrophy and blindness. The pus which forms in the orbit can either break through the palpebral fissure or at a point along the lower lid. If the eye itself is involved, panophthalmitis develops In 4 cases out of 26 which Feuer[1] collected the malady ended fatally by extension to the meninges. The path of infection from the diseased teeth to the orbit can take place through the vessels either along the anterior surface of the superior maxilla, and this is the more frequent form, or through the intermediary of the antrum. The primary infection of the teeth or of its periosteum is often comparatively mild or the condition may develop after the extraction of a tooth. Following the extraction of the tooth, orbital inflammation has developed after from two to ten days had elapsed. The loss of sight can be explained by two possible pathological processes: (1) thrombosis of the central retinal vein by extension of a thrombophlebitis from the ophthalmic veins in the orbit; or (2) an infectious process in the orbital tissue produces a thrombosis of the central vein or possibly of both central vessels. The arrest of the circulation leads to rapid blindness. The ophthalmoscopic condition is a varying one. Frequently the retinal arteries are narrow with slight blurring of the disc margins, a mild papillitis with distended veins, and a few hemorrhages. In other cases, notwithstanding complete blindness, the ophthalmoscopic findings are negative. In some of the cases the ophthalmoscopic picture is that of a pronounced retinal ischemia. The probable cause of the thrombosis of the central retinal artery in these cases is a bacterial disease of the vessel wall, though an increase in the orbital pressure may be of importance. The absence of symptoms of congestion suggests that the blindness is not due to a thrombosis of the trunk of the central vein, though it has been shown that an obstruction which affects the artery as well as the vein prevents the formation of the ophthalmoscopic picture of congestion. The writer[1] observed a case of alveolar periostitis from a tooth infection which was followed by blindness. The only orbital symptoms consisted in periostitis of the floor of the orbit and the ophthalmoscope showed optic atrophy with normal vessels, three weeks after the tooth had been filled. Cavernous sinus thrombosis has arisen from alveolar periostitis following extraction of a tooth; the infection was carried through the pterygoid venous plexus.

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[1] Cited from Groenouw.

The second form of infection which is derived from the teeth is a **chronic sepsis;** this is held responsible for a number of intraocular inflammations. In recent years great advances have been made in recognizing the rôle which tooth infections play in producing obscure infections. The X-ray has been a potent factor in establishing this condition as the teeth may be apparently normal to inspection or as far as symptoms are concerned.

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[1] Knapp, Archives of Ophthal., Vol. XXXV., p. 524.

W. Lang[1] was the first to draw attention to this association. This author says that his attention to oral sepsis was drawn by the observation of William Hern, that pyorrhœa alveolaris is a cause of acne rosacea. He has examined the gums in all cases of inflammatory infections of the eye where the ordinary causes were absent, and believes that pyorrhœa can cause an inflammation of any part of the eye. Out of 10,000 private cases, 215 cases of eye inflammation were due to sepsis, while 168 were attributable to other causes. Of the 215, 139 were due to pyorrhœa. Of the 215 septic cases, the iris was the part of the eye affected 87 times, the ciliary body 79, the choroid 68, the retina 28, the sclerotic 20, the lens 14, the cornea 12 times, and there were 3 cases of detachment of the retina and 4 of optic neuritis or atrophy.

The reason that many cases of pyorrhœa are not associated with septic lesions elsewhere in the body, Lang believes is due to the fact that there is a protecting barrier which prevents the dissemination of the toxin, and that the pyorrhœa with its consecutive complications is due to the crowning of the teeth and the building of bridges, which can not be kept clean. If the protecting barrier is broken down and the eyes should become affected by an inflammation which may destroy sight, it is then too late to do anything beyond removing the teeth.

In infection from teeth Goulden[2] finds that two conditions are possible: (1) abscess in connection with a single tooth; (2) pyorrhœa in which there is a widespread infection of the tooth sockets. This sepsis need not be very severe and the condition of the mouth need not be foul, so that a superficial examination may reveal nothing abnormal, though on expert investigation the tooth sockets are infected. Sepsis in connection with the teeth is the most common cause.

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[1] Lang, Lancet, May 17, 1913, The Influence of Chronic Sepsis upon Eye Diseases.

[2] Goulden, R. L. O. H., 1915.

Lawford[1] accepts the dependence of certain cases of mild chronic irido-cyclitis upon pyorrhœa. The objection to this theory which is raised by many is the frequency of pyorrhœa and the infrequency of the eye complication. Lawford is less inclined to regard scleritis and sclerokeratitis as depending upon this cause, and careful treatment of the mouth has failed to exert any favorable action upon the ocular condition and vaccine treatment has been generally disappointing. He believes that some forms of localized exudative choroiditis may be one of the consequences of pyorrhœa, though he acknowledges that the condition frequently occurs in patients who have no signs of Riggs' disease or of septic diseases in the mouth. He does not believe that the disseminate choroidal disease has anything to do with oral sepsis.

The X-ray is of no value in pyorrhœa. It is of value in determining pus cavities in connection with the roots of the teeth. The X-ray demonstrates whether the root canal has been properly filled to its very apex. The diagnosis of pyorrhœa is generally not difficult. The retraction of the gums and the presence of a cavity between the tooth and the gums out of which frequently pus can be expressed, show the nature of the condition.

H. L. Ulrich[2] finds that a little over 68 per cent. of all artificially devitalized teeth were found with apical abscesses, and the total number of abscesses, on 1350 dead teeth, including those found on pulpless teeth due to caries or accident or to proximal fillings, was 83 per cent. He believes that the apical abscess is merely another evidence of streptococcal focal disease, and is hematogenous in origin. At the time of devitalization of a tooth an apical trauma results which forms a suitable medium for the streptococcal infection. This, he believes, is the explanation of all apical abscesses with the probable except on of those capping pulpless roots following caries. His suggestion, that these foci are not primarily dental but hematogenous in origin, has not been accepted, as many regard these abscesses as due to errors in dental technique, particularly poor root-canal filling. Bacteriological examination of cases of peridental infections[1] has shown that the streptococcus viridans is present in chronic dental abscesses in pure culture, and also in pyorrhœa. Hemolytic streptococcus is absent. These organisms have never been found in healthy teeth. The source of the infection is probably explained by the fact that we constantly find these streptococci in the oral mucous membrane and in the saliva. Henrici has attempted to demonstrate the pathogenecity of dental streptococci by complement fixation tests, which has been done by Besredka, Hastings, and others, and concludes that the results have only been negative, because these patients do not produce antibodies in sufficient quantity to be demonstrated by the method of complement-fixation.

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[1] Lawford, Proc. Royal Soc. Med. Ophth. Sec., p. 121, 1913.

[2] Ulrich, The Blind Dental Abscess, J. A. M. A., Nov. 6, 1915.

In cases with chronic arthritis both the tonsils and teeth are often affected. It is questionable whether the infection which is produced by pyorrhœa is different from that produced by dental abscesses, as the same organism is found in both. There seems to be a relationship between the diseased tonsils, the oral foci and the arthritis. Thus, in those suffering from arthritis the rheumatic symptoms are acutely increased by tonsillectomy, pyorrhœal treatment or by vaccines. Excessive pyorrhœal treatment has not infrequently produced tonsillitis. There can be no question that this oral infection either in the form of apical abscesses or pyorrhœa is frequently the sole cause of arthritis, of rheumatic affections of the muscles, joints and nerves. It is very important that the infected foci must be completely extirpated. It is not enough that the mouth and tonsils appear well from the outside. Even with the aid of the X-ray and careful exploration all foci about the teeth are still difficult to recognize; in fact it is a common experience that the majority of dental abscesses give no clinical sign of their existence. The teeth are not sore, there is no swelling, or soft spot at the root end; it is furthermore amazing to find in well-cared-for mouths how much pyorrhœa does exist without being evident except to painstaking exploration. In the treatment the important feature is to eliminate the local foci and unless these are removed, it can hardly be expected that a vaccine will give any permanent relief.

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[1] Hartzell, Journ. Nat'l Dental Ass'n, November, 1915

Chronic septic foci frequently exert a baneful influence on eyes that have recently been operated upon. Various grades of irido-cyclitis which, in the writer's experience, developed in the second week after an uneventful cataract extraction have been apparently due to tooth infection.

A further very important connection between the teeth and the eyes is a reflex neuralgia which localizes itself either in the eye or back of the eye, and for which the patients come with asthenopic symptoms or to have their glasses changed, without improvement; then a visit to the dentist and the discovery of a defective tooth, and its proper treatment gives prompt relief to the eye symptoms.

The communications of Wm. Hunter,[1] according to S. Marx White,[2] appear to be the first ones to have drawn attention to the importance of oral sepsis. Renewed interest has been aroused by the use of X-ray, which shows the presence or absence of a filling material in the dental pulp cavity and changes occurring in the bone about the teeth. Thus alveolar abscesses have been found in patients in whom such changes were not suspected and could not be demonstrated by earlier methods. These blind alveolar abscesses are remarkably free from local symptoms and signs.

White divides infections from the teeth in the following groups: (1) infections about the teeth, giving the so-called pyorrhœal pocket; (2) abscesses and necroses in the jaw, usually at the tips of the roots with or without sinus formation.

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[1] Hunter, B. M. J., July, 1900; November, 1904.

[2] White, N. Y. State Journ. Med., Vol. XV, No. 12, p. 477.

The importance of these two groups is without question. The recent findings of the endamœba in pyorrhœa gives a new aspect to this group. It is still uncertain whether the endamœba is pathogenic and whether it can form distant infections; knowledge concerning alveolar abscesses is, however, much more definite. In 162 cases the streptococcus was found in 150 (White).

Since the introduction of a dental clinic at the Herman Knapp Memorial Eye Hospital two years ago, the relationship of diseased teeth and lesions of the eyes has been carefully gone into. Without in any way coming to definite conclusions in this short time, it has seemed that the results from the complement-fixation test and from vaccine treatment have not been satisfactory; the treatment of pyorrhœal conditions, however, and the elimination of root abscesses seemed in some cases to be followed with results which were very encouraging and suggestive of further investigation.

AUTO-INTOXICATION

The affections of the eyes which follow disturbances of digestion, particularly of the intestines, so-called intestinal auto-intoxication, have within the last few years received a great deal of attention without bringing the subject into anything like a clear state. The involved question of auto-intoxication and the difficulties of its demonstration are the reason for this.

The relationship between eye diseases and enterogenous toxemia is necessarily a complex one, as our laboratory tests for putrefaction are not altogether trustworthy. This connection was perhaps first suggested by Elschnig[1] in 1905. In his cases indican was found in the urine, which he regards as a sign of perverted metabolism. The regulation of the diet and the administration of intestinal antiseptics improved the eye condition in his cases. Investigations by others soon showed that the single symptom of indican in the urine is no reliable sign of auto-intoxication. Signorine[1] examined 53 cases of phlyctenular keratoconjunctivitis and was not able to demonstrate any sign of an enterogenic auto-intoxication, and he believes that at the present time we must be more or less skeptical in regard to this etiology. de Schweinitz[2] gave the result of a careful examination of the urine, of the feces and of the gastric contents in eye patients. He believes that the clinical picture of the cases of autointoxication is not characteristic. At the same time, in the course of certain uveal diseases whose etiology is obscure, auto-intoxication seems to exert a certain influence. Particularly, relapses seem to occur by the neglect of dietary rules. Flemming, at the above meeting, stated that he found in a large series of urinary examinations that the amount of indican in the urine in the same patient varies enormously at various times of the day. It may be at one time very high, and at the next period very low.

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[1] Elschnig, Kl. M. f. A., November, 1905.

In the International Medical Congress, London, 1913, de Schweinitz states that every case of uveitis is of septic or toxic origin, and that the term "rheumatic iritis" should be discarded. Acute articular rheumatism is very rarely a cause of uveitis. While in cases of iritis a previous history of chronic muscular rheumatism or chronic articular rheumatism is frequently given, the iritis is due to auto-intoxication and has nothing to do with rheumatism. It is important in these cases to examine the urine and the metabolism. The absence of indican is not a proof that intestinal auto-intoxication does not exist. In a recent article[3] this author says: "Although there is no proof that any toxic substance elaborated within the tissues in the course of a so-called gastro-intestinal auto-intoxication has of itself by its toxic properties produced a uveitis, we do know that intestinal putrefaction depends upon the activity of bacteria upon the food-stuffs in the intestines, and there is good reason to believe that these bacteria or their toxic products may be the cause of an inflammation of the uveal tract, exactly in the same way as bacteria arising from focal infections elsewhere in the body have a similar influence, and therefore gastro-intestinal intoxications are of great importance in their etiological relationship to uveal tract disorders."

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[1] Signorine, Arch. d'Ottalm. Vol. XX, 1913.

[2] de Schweinitz, Sec. on Ophth., N. Y. Acad. Med., Archives of Ophth., 1912.

[3] De Schweinitz, Ophthalmic Record, December, 1915.

Lawford,[1] in a discussion on alimentary toxemia, believes that alimentary toxemia is a contributory factor—if not the sole agent in causing eye lesions. He believes that auto-intoxication from the alimentary tract may be regarded as a good working hypothesis and that it helps to explain the clinical appearances, particularly of diseases of the sclero-cornea and of the uveal tract.

The etiology of these toxic conditions has been remarkably aided by the study of focal infections, a subject to which a great deal of attention is at present being given and one requiring the coöperation of several branches of medicine. Focal infections, in general, may be found in the following locations: (1) the nasal accessory cavities; (2) the middle ear and antrum; (3) tonsils; (4) alveolar processes; (5) the genito-urinary tract, usually the prostate and seminal vesicles in the male and the uterine mucosa in the female; (6) gastro-intestinal tract, including the gall bladder and the appendix. These individual foci will be taken up in their respective chapters.

As rheumatic affections of joints and muscles are unquestionably toxic, it is fitting to consider the so-called rheumatic affections of the eye at this point. The diagnosis of rheumatism in the eye is undergoing modification and restriction. By some authors it is being replaced by the term "toxemic." In acute articular rheumatism, unquestionably an infectious disease, a form of cryptogenetic sepsis, eye complications are examples of metastatic inflammation. They are observed not only in the severe cases and in those complicated with cardiac lesions, but also in the mild cases. The eye disease which is most frequently present is an inflammation of the uvea, particularly iritis, which may occur at the same time with the joint affection or alternate with it. Episcleritis, tenonitis, interstitial keratitis, optic neuritis and paralysis of the external ocular muscles have also been described. Poynton and Paine[1] state that rheumatic fever causes endopericarditis, arthritis, pleurisy and subcutaneous nodules. Rheumatic iritis is rare, 1 in 270 cases (Macrae). A boy at nine developed active rheumatism, *i.e.*, arthritis, pericarditis and subcutaneous nodules. Death from pericarditis. One of the rabbits inoculated intravenously with this pericardial fluid developed iritis. They mention a case published by Forster,[2] of a girl twelve and one-half years of age, with chill, tonsillitis, arthritis, chorea, iritis in the right eye and endocarditis.

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[1] Lawford, Proceed. Roy. Soc. Med., May, 1913, p. 121.

Chronic rheumatism can not be defined at the present time, while rheumatism is usually associated with three symptoms: (1) its development after exposure to cold; (2) pain; (3) that certain forms of tissue, muscles, fascia, tendons, ligaments and joints are predisposed, with the advance in medical knowledge its domain has become restricted. Many of the symptoms in infectious diseases were formerly classed as rheumatic. It is not possible to separate muscular rheumatism and chronic articular rheumatism. The rheumatic pains frequently are indefinite and in chronic articular rheumatism the pain does not seem to be limited to joints, but involves the tendons and the fascia. There are unquestionably affections of the eye which result from exposure to cold. They are, however, not necessarily rheumatic unless we find in the body other evidences of a rheumatic diathesis. It is, moreover, very frequent that in taking a history of a patient with eye disease, ill-defined pains in muscles and joints will be obtained. Many cases of iritis are surely associated with rheumatism, wherein the condition seems to depend upon changes in the weather and are often associated with ill-defined sensations in the joints or in the muscles.

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[1] Poynton and Paine, Rheumatic Iritis, T. O. S., 1903.

[2] B. M. J., 7, III, 1903.

We can not, however, give up the rheumatic tendency entirely and from a therapeutic standpoint the study of the eye patient's diathesis[1] is of importance. The affections characterized by organic hyperacidity formerly were called arthritic, though many of these patients show no joint affections, but rather a disturbed function of the liver. They have a common symptom in an increase of the normal acidity of the urine. Arthritism does not exist if the liver is normal, and the condition can practically be termed hepatism. Arthritism includes rheumatism, gout and dermatosis. According to Giraud, among the causes of eye diseases the arthritic diathesis occupies the first place, second syphilis, third tuberculosis. The main characteristic of these arthritic conditions consists in the uselessness of local application while general treatment is of great avail. The general treatment means the regulation of diet and in exercise, with the internal administration of alkalies, of sodium salicylate and the regulation of the liver function. These patients will often show manifestations of a dry and itching skin, of eczema or of some skin disease.

It may be of interest here to review a number of statistics on the etiology of iritis. Nettleship[2] investigated 71 cases of iritis. Of these 30 were syphilitic, 23 seemed to be rheumatic, though 6 of these were due to gout and 1 to gonorrhœa. Gutmann,[3] from the Michel Clinic, in 150 cases of primary iritis, found syphilis in 32 per cent., tuberculosis 27 per cent., chronic nephritis 5 per cent., arteriosclerosis 13 per cent., rheumatism and gonorrhœa

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[1] Giraud, L'Oeil Diathésique, Relation de la Diathése avec les Affections des Organes de la Vision, and Ramsay, Diathesis and Ocular Diseases, Bailliere, Tyndall & Cox, London, 1909.

[2] Nettleship, B. M. J., 1876.

[3] Gutmann, B. m. W., No. 42, p. 1671, 1905.

3 per cent. each; combined causes 4 per cent., unknown 12 per cent. Jennings and Hill[1] tabulated 500 cases of iritis in which syphilis is the cause in 61.4 per cent., rheumatism 25.4 per cent., gonorrhœa 5.2 per cent., while other systemic disorders are occasional causes.

Irons and Brown[2] found that the causes of 100 cases of iritis were as follows: syphilis 23; gonorrhœa 9; tuberculosis 8; dental infection (closed) 18; tonsils (excluding superficial crypts) 16; nasal sinus 3; genito-urinary tract 3; other suppurations 2; no cause 1; combined infection 17. In the last group: syphilis 8; gonorrhœa 9; tuberculosis 7; dental 8; tonsils 13; nasal sinus 5; genito-urinary 3. This represents an exhaustive study of every etiological factor by all available methods; chemical, serological, bacteriological, X-ray, etc. It is interesting to compare these statistics, showing changes in the medical viewpoint and the way along which more accurate information on etiology is obtainable.

As aids to diagnosis, the following examinations can be undertaken. Mayou[3] finds that there are three kinds of bacteria which usually cause cyclitis and that these are usually present in the aqueous humor. Among 30 cases he finds tubercle bacillus in 13, staphylococcus in 10, and the spirochete in 5. He suggests as a routine method of examination first the Wassermann test, second the von Pirquet test, then paracentesis and examination of the aqueous. Of the 10 cases treated with staphylococcus vaccine, generally prepared from organisms obtained from the anterior chamber, 6 cleared up and 4 improved. To examine the aqueous humor, Harman[4] has devised a hypodermic needle with sharp spear-shaped head which when applied to a lacrymal syringe can be introduced through the cornea and the aqueous withdrawn. Browning[5] recommends the frequent routine examination of the feces and urine, particularly when there is a history of colitis obtainable. He has been able to find on culturing pneumococcus and streptococcus. Vaccines were obtained and treatment followed in some cases with benefit, in others without. The examination of the urine has shown a number of causes, particularly of keratitis, to which Lawson has drawn attention, to be associated with the presence of the bacterium coli in the urine. After the recovery of this organism and the use of the vaccine, the eye condition rapidly improved.

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[1] Jennings and Hill, Ophthalmology, Vol. VI, p. 52.

[2] Irons and Brown, Am. Ophth. Soc., 1916, J. A. M. A.

[3] Mayou, B. M. J., p. 1324, 1911.

[4] Harman, Ophthalmic Review, December, 1908.

[5] Browning, Ophthalmic Review, Vol. XXXII.

The following are the diseases of the eye which seem to be auto-toxemic in origin; at least the arthritic, uric-acid, rheumatic or auto-toxic nature is shown by the recovery of the patient after general treatment is instituted, consisting principally in a restriction of protein diet, regulation of the bowels and exercise. A conjunctivitis which is not infrequent is more or less characteristic, in that the symptoms are out of proportion to the degree of local disturbance. The patients particularly complain of a dryness, of a sticking of the lids to the eyeball. They sometimes present clear blisters along the inner margin of the eyelids. The local treatment in these cases should be mild—boric acid and hot compresses particularly.

Cases which in the writer's experience were distinctly auto-toxic and which yielded readily to dietary measures, where certain forms of scleritis and marginal keratitis; the ocular treatment was not nearly as efficacious in effecting a cure as a calomel purge and a meat-free diet and the characteristic relapses were best avoided by adherence to a strict diet. The writer has notes of two cases of spasm of the sympathetic nerve in women of about thirty-five years of age, with symptoms of a monolateral irritation of the sympathetic nerve producing exophthalmos and retraction of the upper lid which disappeared after the auto-toxemia was corrected.

The **bacterium coli commune** has been brought into etiologic relation to some intraocular affections, and it is one of the organisms to be considered in searching for the cause of obscure infections. The patients give a history of chronic cystitis or recurrent diarrhœa or both. Macleish[1] has observed five cases of keratitis of a vesicular type in whom a pure culture of the bacillus coli was obtained from the urine in each patient and in one case from the aspirated contents of the anterior chamber. The surface is stippled and there are larger blebs; in short a condition resembling dystrophy. The substantia propria is diffusely infiltrated, soggy or striped, or the infiltrations are in patches with occasional deep striations. The patients were all over sixty years of age, and the duration of the keratitis varied from one to seven years. Great improvement in the corneal condition resulted from treatment with an autogenous vaccine, urinary antiseptics and buttermilk. Lawson[2] has reported a case of optic neuritis with exudates and hemorrhages as an example of bacillus coli toxæmia and a case of vesicular keratitis which cleared on the use of b. coli vaccine.

THE LIVER

The most frequent symptom of diseases of the liver and of the bile passages is **jaundice,** and as is well known, it frequently affects the conjunctiva. In jaundice retinal hemorrhages sometimes occur. They are an evidence of a general hemorrhagic tendency which is distributed among a number of organs, and are not necessarily of evil omen, as they may be present in quite harmless catarrhal jaundice.

In chronic jaundice **xanthelasma** occurs in the eye lids as well as in other parts of the skin. A characteristic is its symmetrical development. It is a benign tumor which grows but slowly and requires removal only for cosmetic reasons. A functional disturbance of the liver is generally present.

[1] Macleish, Arch. of Ophth., Vol. XLIV, p. 403, 1915.

[2] Lawson, T. O. S., XXXI, p. 27, 1912.

Yellow vision (xanthopsia) of the jaundiced is an old symptom, but is very unusual. The patients sometimes complain of this symptom; in other cases it is demonstrated on examining the color sense. Xanthopsia is explained by a yellowish discoloration of the refractive media of the eye with bile pigments.

In addition to the hemorrhages in jaundice, white areas have been observed in the retina, and neuro-retinitis. Changes in the pigment epithelium have been described in jaundice with night-blindness. The eye grounds showed a marked paneling with small dots and pigment foci. This condition which has been called **hepatic ophthalmia,** has been found to consist in a cellular infiltration of the choroid and in changes in the retina, particularly in the pigment epithelium—an œdema of the retina and of the optic nerve with inflammatory changes in the vessel walls.

Night-blindness as a symptom in those suffering from diseases of the liver is a well-recognized condition, and there is no question that there is a distinct connection between the two. It is, however, not true that every patient with night-blindness presents signs of jaundice. The night-blindness occurs in a variety of diseases of the liver when they are accompanied with jaundice, and it usually appears a certain length of time after the jaundice. The ophthalmoscopic picture is negative, though changes in the pigment epithelium have been reported. This night-blindness has been improved by treatment in a dark room. It is not always a sign of bad prognosis, and it usually occurs only in severe cases of jaundice.

A complication of night-blindness is **epithelial xerosis of the conjunctiva.** The explanation for the night-blindness in jaundice may be found in the general nutritional disturbance through lack of bile or through changes in the blood, as it is known that the bile acids cause a solution of the visual purple.

Ishihara[1] states that xerosis of the conjunctiva and night-blindness occur particularly in children who are suffering from the general disturbances of nutrition. Though they may not terminate fatally through the addition of keratomalacia, sight is apt to be lost in a short time. The author believes that cod-liver oil is an excellent remedy and almost a specific in this condition. It is supposed to have helped cases even of keratomalacia as long as the corneal process had not gone too far; the conjunctiva regains its moist condition, and the corneal ulcer is arrested and regenerates. Liver is also of value in the treatment of this condition.

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[1] Ishihara, K. M. f. A., p 596, Vol. LI.

Mori believes that poverty in fat in food or insufficient absorption of fat from the intestines is the injurious moment in xerosis and keratomalacia. As cod liver oil is an easily absorbable fat, its value is explained. In two cases which this author examined, he found that the amount of fat contained in the blood in night-blindness and xerosis was more or less diminished. It is entirely independent of the general nutrition of the patient and of the amount of subcutaneous fat. He is convinced that these conditions depend upon a lack of the fatty substances in the blood and it therefore occurs in all conditions of general disturbance of nutrition, lack of fat in the food, diseases of the liver, diarrhœa, etc.

GOUT

In the etiology of eye diseases gout plays a definite though restricted rôle. As the interpretation of gout is not very definite, it has not always been easy to confirm the etiological factor. Gouty individuals are often the subjects of obstinate conjunctivitis. The other diseases of the eye are keratitis, scleritis, iritis and glaucoma. It is, of course, impossible to demonstrate either anatomically or chemically that these diseases are caused by gout. The eye disease, however, may be the first manifestation of gout. As the urate foci are found frequently in tissues poor in blood vessels, a localization in the cornea and the sclera is to be expected. The lesion may extend to the other tissues. In the anterior part of the sclera flat or pointed prominences appear with marked hyperemia which may resemble tophi. Wagenmann found in an eye a grayish prominence near the papilla in addition to a typical gouty focus anterior on the sclera.

Krückmann[1] describes typical ocular paroxysms in gout: Sudden onset of pain at night with relief during the day, swelling of lids, marked chemosis, hyperemia of eyeball. The lids are remarkably tender, cornea dull, posterior synechia. The external symptoms are out of proportion to the internal inflammation. The prognosis is good, only slight functional disturbance remains. This may be the first manifestation of gout.

Iritis and cyclitis usually appear after a gouty attack in some other part of the body, accompanied with intense headache, and are very obstinate. Many cases are not different from those after any articular affection. As the ocular complications are, however, severe and as they do not necessarily present anything characteristic, the proper recognition and treatment of these cases depends upon the general examination of the patients. According to Lichtwitz,[2] the demonstration of uric acid in the blood, the presence of other gouty manifestations, particularly with the aid of the Rœntgen picture, the presence of tophi, are all important. Doyne[3] has described small warty transparent excrescences which appear along the pupillary margin of the iris without any sign of inflammation in elderly and gouty subjects, and has given it the name of guttate iritis. Brailey[4] reports the case of a man with gout who suffered from sudden and acute attacks of iridocyclitis. The attacks were characterized by the suddenness of onset, coming on at night, and their speedy decline.

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[1] Krückmann, Med. Klinik, 1910, No. 38.

[2] Lichtwitz, Archives of Ophthalmology, 1914, p. 24.

[3] Doyne, T. O. S., Vol. XXX, pp. 91 and 229.

[4] Brailey, T. O. S., 90.

They resembled the attacks of pure gout which he formerly experienced in his feet and limbs.

ŒSOPHAGUS

In carcinoma of the œsophagus in one-sixth of all cases there was a difference in the pupils. The left pupil was regularly narrower than the right. The lesion is dependent upon a disturbance of the sympathetic nerve.

INTESTINAL WORMS

The ocular complications depend upon the toxic action of these parasites or upon the direct invasion of the eye by parasitic embryos, or, finally, the affections of the eye are secondary to the anæmia caused by the presence of these parasites.

Of the parasites which may wander from the intestinal tract and penetrate the eye, should be mentioned the cysticercus cellulosæ. This worm inhabits the small intestine, its eggs are discharged in the bowel movements and thus can contaminate drinking water and enter the intestinal tract. After leaving the intestinal tract, it is carried by the blood to the various organs of the body and then finally becomes lodged and transformed in a small vesicle (cysticercus). This parasite occurs principally in pigs. In the eye the cysticercus has been observed in the skin of the lids, in the parts about the eye, under the conjunctiva, in the orbit, in the anterior chamber, vitreous or retina. It occurs in certain areas, though careful meat inspection has practically abolished its appearance. The echinococcus is a parasite which occurs in the intestines of the dog. It may develop to a large size and has not been observed in the eyeball, but in the orbit. Filaria enter the body with the drinking water. They have been observed under the conjunctiva, in the orbit and in the anterior chamber. Intestinal parasites, particularly the ankylostomum and bothriocephalus, may produce severe secondary anemias.

IX. ANÆMIA

Chlorosis.—In addition to functional disturbances, there are a number of organic diseases of the eye which have been brought into relationship with chlorosis. Thus, there is œdema of the lids which is apt to be most marked early in the morning and may suggest nephritis. The conjunctiva sometimes shows hyperemia or a catarrhal condition with its symptoms of pressure and a sandy feeling. The retinal vessels naturally have attracted the greatest interest in this condition, though in only a small proportion do they present any anomaly. In this connection the great physiological variations in the appearance of the eye ground must not be forgotten. With the upright image it is possible sometimes to detect a pallor of the eye ground and a somewhat lighter color of the blood. On the other hand, in some cases a congestion of the disc and of the retinal veins was observed.

Uhthoff[1] has found that the vessels, especially the veins, appear much paler on the disc than elsewhere. Pulsation in the arteries was observed in a few cases.

Retinal hemorrhages and slight inflammatory symptoms in the retina and optic nerve have been described. It is uncertain whether these cases should be classified strictly as belonging to anæmia, and this diagnosis should be made only after the most careful exclusion of other diseases, particularly of the brain or of the kidneys. Pronounced optic neuritis and choked disc have been found associated with chlorosis, and in some of these cases cerebral symptoms—headache, vertigo and vomiting—have preceded or were associated with the optic neuritis. There are also reports of simultaneous paralyses of ocular muscles and of other parts of the body. The characteristic feature of these cases is that they have recovered under the administration of iron. Uhthoff believes that the cases of optic neuritis and choked disc should not be ascribed to chlorosis, especially in the presence of an ocular muscle paralysis, as there are cases of optic neuritis of unknown etiology that get well. Leber finds that retinal hemorrhages are very unusual in chlorosis, and it is a question whether the cases in which they are observed belong to chlorosis rather than to secondary anæmia.

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[1] Uhthoff, quoted from Groenouw, p. 291.

Pagenstecher,[1] in 246 cases of chlorosis from the Hamburg Medical Clinic, found retinal hemorrhages in only 3. He further has observed that in these cases the intracranial pressure has been found raised, as shown by lumbar puncture. This is of particular interest and importance in explaining the choked disc.

Pernicious Anæmia.—In severe anæmias multiple hemorrhages and small white degenerative foci are observed in the retina. These changes in the retina occur in a variety of forms of anæmia, in the idiopathic pernicious and the secondary anæmias. They are important from a diagnostic standpoint, from the great regularity with which they occur. This is particularly true in the progressive pernicious type of anæmia, where they occur in 50 per cent. of the cases; some claim that in the acme of the disease they are practically constant. The hemorrhages resemble in form those that occur in septic and hemorrhagic processes. At first they are not particularly numerous, isolated, scattered over various parts of the eye ground, usually in the neighborhood of the disc, along the large vessels. They may quickly disappear, while fresh hemorrhages occur in different places. Sometimes when they are of a round form, a paler yellowish or a grayish-white center appears, and they resemble a white spot surrounded by a red margin.

In addition to the hemorrhages, there may be small white dots which apparently do not develop from hemorrhages and are probably ganglioform or varicose nerve fibres which are also seen in thrombosis of the central vein and in nephritic retinitis.

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[1] Pagenstecher, Archiv f. Augenh'lk., Vol. LII, p. 237, 1906.

Occasionally there are white spots without hemorrhages and it is possible to have a picture which resembles albuminuric retinitis including the star-shaped figure at the macula. In pronounced cases there is a slight blurring of the edges of the disc and the surrounding retina, probably due to an œdema; the disc is pale and the retinal vessels are narrowed.

According to Heine,[1] in pernicious anæmia the optic nerve is pale in both eyes, almost white and atrophic, the margins are blurred on account of the blurring of the surrounding retina. The vessels, both arteries and veins, are pale and consequently not as distinct on the disc as in the retina, and appear to be broken off at the margin of the disc. This last feature occurs when the hemoglobin sinks below 40 per cent. In high-grade anæmia the blood vessels are not to be recognized; their contents appears to be watery. Small hemorrhages followed after a few days by multiple ones, then very large extravasations of blood, are very characteristic and indicate a serious prognosis.

Leber[2] states that we do not understand how the retinal changes in pernicious anæmia are produced. Toxins soluble in the blood may injure the endothelium of the vessel wall, or a slowing of the circulation or an occlusion of the smaller vessels may permit diapedesis.

A similar ophthalmoscopic picture occurs in the anæmia following intestinal parasites, cancer and tuberculosis. Thus, anchylostoma duodenale has caused severe anæmias, also the botriocephalus. The eye grounds in these cases showed pallor, thin vessels, pale blood columns, arterial pulse and hemorrhages. The hemorrhages are probably due to the action of toxins, as they occur after the stage where the parasites have produced a high-grade anæmia. Secondary anæmia in the cachexias following malignant tumors frequently produce retinal complications, and certain authors speak of a cachectic retinitis. Pick,[1] in 1901, found it present in 30 per cent. of the cases in malignant tumors, though believes they are much more frequent, between 60 and 70 per cent. The cases reported by this author were all carcinomas of the stomach and liver. These cases, according to Pick, differ from those previously described, through the greater frequency of white foci, while hemorrhages are less noticeable and may be absent. These white areas are situated near the disc, usually at the site of bifurcation of the vessels, are grayish-white, diffuse, and vary in size. The hemorrhages have a white center. They are due to toxin action, according to Leber, as hemolytic substances have been found in malignant tumors. These changes may occur in any severe anæmias following cirrhosis of the liver, ulcer of the stomach, tuberculosis of the intestines and nephritis. Pick[2] draws attention to relatively slight changes occurring in chronic nephritis, isolated grayish areas and small hemorrhages in the retina which he believes are due to anæmia and differ from true albuminuric retinitis.

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[1] Heine, Augenuntersuchung, etc., p. 76.

[2] Leber, Graefe-Saemisch-Hess, II ed.

Amaurosis after Loss of Blood.—Retinal changes similar to those just described occur in severe anæmias following profuse and repeated hemorrhages. These have been reported particularly after hemorrhages of the stomach, of the uterus, and from the nose.

Fries,[3] in 1876, studied the published cases and found the hemorrhages took place from the gastro-intestinal tract in 36 per cent. of the cases, from the uterus in 25 per cent., artificial blood letting 25 per cent., epistaxis 7 per cent., from wounds 5 per cent., hemoptysis and urethra 1 per cent. With the recurrence of the hemorrhage the vision may be progressively affected, or with each attack blindness may result with subsequent improvement. Groenouw states that it is important to remember that this disaster occurs in persons who were ill before the hemorrhage, and that healthy individuals who have suddenly lost a great deal of blood, as soldiers in battle, practically never suffer from loss of sight.

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[1] Pick, Kl. M. f. A., p. 177, 1901.

[2] Quoted from Groenouw.

[3] Fries, Kl. M. f. A., XIV, Inaug. Diss., quoted from Groenouw.

The patients are usually over forty years of age. Both eyes are generally equally affected, in 10 to 15 per cent. one eye alone was affected. Vision is impaired in 25 per cent. of the cases at the time of the hemorrhage, in an additional 20 per cent. within the first twelve hours, and in more than half at a later period, usually between the third and sixth day, exceptionally after eighteen to twenty-one days. With the hemorrhage the patient suffers from faintness, headache, depression, palpitation, severe pain in the occiput and neck. When the vision becomes affected, the patient has usually recovered from the general prostration. Blindness lasts for a variable period, from fifteen minutes to a day, and the improvement is very slow if it takes place at all. In one-half of the patients sight does not return; in one-third, improvement occurred; and in one-fifth, vision was restored though with field defects. The examination of the eyes has shown dilated and immobile pupils, a blurred disc, and a gray and clouded retina. The disc becomes white and the arteries small.

Gessner[1] reported on a severe hemorrhage occurring three weeks after labor, which led to complete blindness in two days. Three days after the onset of the loss of vision the examination with the ophthalmoscope showed a pronounced choked disc.

Hirschberg[2] observed a case which was examined with the ophthalmoscope even before the onset of the eye changes. Three days after the loss of blood the left disc showed a white clouding, the right, distinct inflammation. Five days later, neuro-retinitis was present in both eyes, right more marked than left. On the tenth day the right eye was blind. Two days later the neuro-retinitis began to retrograde and on the fourteenth optic atrophy was present in the right, while the left eye was nearly normal. The course of the changes was unusually rapid.

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[1] Gessner, Arch. f. Augenh'lk., 1889.

[2] Hirschberg, Arch. f. klin. Med., Vol. IV, 1881.

Leber[1] speaks of small hemorrhagic or degenerative retinal areas which are present in the beginning stage without marked involvement of sight, with œdema and changes in the retinal tissue, just as in the secondary anæmias in chronic diseases and in cancer cachexia (retinitis cachecticorum). He believes that a pernicious anæmia is the primary cause for the retinal changes in some of these cases and that both infectious and toxic agencies are active. This explanation, of course, does not apply to many of the cases such as hemorrhage after gastric ulcer, abortion or venesection.

In these cases, if the anæmia is severe enough, similar changes can occur in the retina, circulatory disturbances, loss of sight, multiple retinal hemorrhages and small white degenerative areas. It is possible, in Leber's opinion, that the changes in these cases are produced not only through a direct loss of blood, but through the retardation of the circulation which, in turn, can produce œdema and multiple hemorrhages in the retina.

Numerous theories have been advanced to explain the pathological process. It seems probable, and Holden's[2] investigations have shown, that the process is a peripheric one and the cause must lie in some product which is formed in the restoration of the lost blood and which is toxic under certain conditions.

Leukemia.—Changes in the eye ground have been observed in all forms of chronic leukemia which are the result of the enormous increase in the number of leukocytes and a disturbance of the circulation, due to the changed condition of the blood. The large number of leukocytes in the blood and the hyperemia of the retinal vessels cause a changed appearance of the vessels and of the hemorrhages, and under certain circumstances give rise to a strikingly pale, rather yellowish color of the eye ground. The veins, which are usually enormously distended, present a pale pinkish color. The arteries which are not dilated are usually of an orange-yellow. The differences between these two vessels in color is slight. The color of the hemorrhages is frequently pale. The choroidal vessels, if they are visible, and the entire eye ground are pale orange-colored or yellow. This, however, is present in only a small proportion of the cases. The unusual color of the eye ground depends, in Leber's opinion, upon a slight pigmentation of the retinal epithelium, so that the lighter color of the blood in the choroid becomes visible.

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[1] Leber, l. c., p. 1016.

[2] Holden, Arch. of Ophth., XXVIII, 1899.

Hyperemia is always present, evidenced principally in a dilatation and a tortuosity of the veins. The arteries are not usually affected. The veins may be enlarged to four or five times the size of the arteries. When they are so distended, it sometimes is possible to observe the circulation of the blood with the ophthalmoscope. This has been done by Grunert.[1]

The circumscribed changes occur in the form of hemorrhages, sometimes white areas with or without hemorrhagic margins. Simple hemorrhages are very frequently present, even without hyperemia and retinitis. They are present in large number and present a characteristic change in color. They may give rise to large vitreous hemorrhages which entirely mask the eye ground, and result in secondary glaucoma, detachment, iritis, etc. Hemorrhages may also occur in the choroid. Hemorrhages of the conjunctiva have been observed.

In addition to hemorrhages, in most cases of pronounced retinitis, there are circumscribed white areas which are surrounded with a hemorrhagic margin or accompanied with some form of hemorrhage. These leukemic foci are yellowish-white, round, distinctly prominent, and surrounded with a hemorrhagic margin. In many cases they appear principally in the anterior parts of the eye ground, about the equator, as is shown in an illustration of Jessop's.[1]

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[1] Grunert, Centralblatt f. p. Augenh'lk., 1901.

Small whitish spots without hemorrhages have been observed in large numbers in addition to the previously described areas. Sight is usually not affected, unless the macular region is involved. This retinitis is not a constant symptom of leukemia, though like in albuminuric retinitis the condition of the eye ground may be the first to draw attention to the general affection.

The frequency of retinal complications has not been determined. Possibly they are present in one-fourth to one-fifth of the cases. A statistic of Meyer in 1889, shows that out of 20 cases retinal hemorrhages were observed in 8. In acute leukemia retinal changes are not as frequent as in the chronic form. The condition is always bilateral. Other eye changes in leukemia consist in hemorrhages in the lids, conjunctiva, and in the anterior chamber and vitreous.

An important complication of leukemia is the bilateral formation of lymphomatous tumors of the lids and of the orbit. They frequently involve the lacrymal gland. The skin of the lid is tense, shiny, with dilated veins, not adherent to the underlying tumors. These lymphomata occur either as small nodules or as a large lobular elastic mass. They are generally symmetrical, which is characteristic. In some cases they seem to appear exclusively in the eyelids and no where else in the rest of the body. If they are situated within the orbit, they cause the symptoms of an orbital tumor. Treatment in most cases is of no avail. Operations are contra-indicated, on account of the tendency to hemorrhages. A not infrequent complication is associated deafness.

Pseudo-leukemia.—In the closely associated pseudo-leukemia, in which there is no increase of white blood corpuscles, similar retinal changes have been observed. The tumors occurring in the lids and in the orbit in this condition are often complicated with changes in the retina and in the optic nerve.

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[1] Jessop, T. O. S., 1898.

Retinal changes occur in all forms of leukemia, especially in the chronic varieties where the spleen is enormously enlarged, though it may occur in cases where the lymphatic glands and the bone marrow are chiefly affected. Though the retinal conditions were formerly described only in the chronic cases, a number of cases have been reported where they appeared in acute leukemia. Elschnig[1] found characteristic changes in acute leukemia, consisting in enormous distention of the veins, of pale color, hemorrhages and cloudy retina. Werner[2] described a lymphoma of the conjunctiva in a woman aged twenty-five, who presented a cushion-like gelatinous mass, formed by the greatly enlarged retrotarsal folds; the tarsal conjunctiva was also gelatinous. He describes the condition under the following headings: (1) secondary involvement of the conjunctiva especially of the upper retrotarsal folds in lymphomatous tumors of the orbit or lids; primarily as (2) pseudotrachoma; (3) small tumors in the bulbar conjunctiva chiefly affecting plica; (4) diffuse lymphoid infiltration like the above case; (5) amyloid degeneration.

The various hemorrhagic conditions, such as **purpura, scurvy** and **hemophilia,** sometimes cause retinal hemorrhages. The vessel walls in these conditions show no change. Most of these cases have occurred in purpura. They were usually severe and terminated fatally. The hemorrhages have no characteristic signs. In scurvy retinal hemorrhages are very unusual. In the few cases in which hemophilia has seemed to be the cause for retinal hemorrhages, the cases have been very severe and the hemorrhages have been massive and relapsing. In some of these cases the condition is probably similar to that found in recurring hemorrhages of adolescence. Scurvy[3] attacks children between six and eighteen months in the cold part of the year. Some features of rickets are generally present, epiphyseal enlargement, and curvature of the bones, and hemorrhages in various parts of the body. The gums may be spongy, though this is not present until the teeth have appeared. In the orbits there may be great effusion into the lids, proptosis, followed later by blood staining of the skin. In infants it may show itself only by a mere line of blood staining at the orbital rim. The larger hemorrhages occurring subperiosteally distend the upper lid, and the blood staining occurs much later.

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[1] Elschnig, Wiener med. Wochensch., p. 1435, 1899.

[2] Werner, T. O. S., 1904.

[3] Spicer, T. O. S., 1892.

Polycythemia (see page 329).—The change in the blood consists in an increase of the number of red blood cells to 7, 10, 14 billions in 1 *cc.* The characteristics of the blood cells are, however, not altered. The general plethora shows itself by a distention of the superficial veins. It is supposed to be due to a functional over-action of the bony medulla. The skin and the mucous membrane are often of a dark red. There may be an occasional albuminuria. A characteristic symptom is severe attacks of pain which are apt to occur at night in the lower extremities. The eye grounds show very markedly distended veins with curious varicose dilatations and occasionally small hemorrhages. Ascher[1] states that the external signs on the part of the eyes in polycythemia are a dark livid hyperemia of the conjunctiva, particularly of the palpebral conjunctiva and a bluish discoloration of the sclera. The hyperemia differs from that found in conjunctivitis by its synchronous development in both eyes without irritation or secretion. The fundus changes consist in the central vessels being twice as large as normal and of a dark red color with a pronounced reflex. The vessels are tortuous and there is usually a decided swelling of the disc. Hemorrhages are not present and vision is unaffected.

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[1] Ascher, K. M. f. A., Vol. LIII, 1914.

X. DISEASES OF THE KIDNEYS

The eye is involved in the course of nephritis in several ways. The most important is **albuminuric retinitis,** which occurs usually in chronic nephritis and then most commonly in the contracted kidney. Albuminuric retinitis is a well-defined condition. The changes in the retina, though varying in degree, are those of an inflammation characterized by hemorrhages, and particularly by processes depending upon fatty degeneration. The retina shows a pronounced **hyperemia** in which the arteries are only slightly involved, and a **sero-fibrinous exudation.** There are cases in which the hyperemia is absent and Leber suggests that these be termed nephritic degeneration of the retina. The retinitis is usually called albuminuric because albuminuria is one of the constant symptoms of nephritis, and its demonstration is a rapid means of diagnosis. The albuminuria, however, has nothing directly to do with the development of the retinitis. It may exceptionally and for a long time be absent. Furthermore, every retinitis in which albuminuria is present is not cased by nephritis, and Leber, therefore, prefers to call the disease a nephritic rather than an albuminuric retinitis.

Nephritic retinitis is never a precursor of the nephritis and occurs only when a distinct functional disturbance of the kidneys has taken place, combined with increased arterial pressure, and in chronic cases with hypertrophy of the left ventricle. It, therefore, belongs in chronic nephritis to a late stage and is not unusually an indicator of a severe, fatal complication. Occasionally it is, however, the first symptom of a disease which has previously run a latent course. In acute nephritis it may occur simultaneously with the first symptom, though even then the fundamental disease has reached a marked development. The reason that the retinitis has been regarded as a precursor of nephritis is on account of the well-known characteristic of the contracted kidney, that it may exist for a long time without causing the patient any symptoms. In these cases albuminuria may occasionally or during the entire course be absent. The presence of a cardiac hypertrophy and repeated urinary examinations will correct this error, except in those cases in which the albumen is absent for a long time or continuously.

True nephritic retinitis can occur in all chronic and acute diseases of the kidney which lead to the development of albuminuria and to the retention of the poisonous or excretory substances, and is consequently absent in others where there is no distinct disturbance of the secretory kidney function. It is not dependent upon the presence of the albuminuria nor upon the presence of œdemas.

It is most frequent in the **contracted kidney,** in the primary form as well as in the chronic form. In this, the quantity of urine is large, of low specific gravity, greenish-yellow color, slight quantity of albumen, no sediment; there is cardiac hypertrophy and tension pulse, no hydrops, running an insidious, latent course. It occurs in general arteriosclerosis, alcoholism, gout and lead poisoning.

The ophthalmologist is well acquainted with those cases where the kidney lesion is quite latent and insidious, and the only general symptoms consist in headache and gastric disturbances. The general condition of the patient has not particularly depreciated but a pale color of the face is noticeable. If the disturbance of vision occurs at this stage, the patient will first consult an oculist. The blood pressure is increased and the left ventricle is hypertrophied. Sudden uræmic attacks are not infrequent, which may be associated with blindness. Death follows in one of these attacks or by cerebral hemorrhage or an inflammation of a serous membrane.

Albuminuric retinitis occurs less frequently in the diffuse chronic parenchymatous form, characterized by marked hydrops, albumen in large quantity, urine usually diminished, of high specific gravity and characteristic sediment. This form frequently passes into the contracted form, which, according to Michel, is always revealed by the ophthalmoscopic changes.

Leber states that the retinitis is not dependent on any particular form of nephritis but results from functional disturbance or loss of the secretory parenchyma, which occurs in the various forms of nephritis.

Thirdly, retinitis occurs in acute nephritis, especially after scarlet fever, which usually gets well. There is marked anasarca, albumen in large quantity, and blood; quantity diminished, of high specific gravity and the sediment contains red blood cells and casts.

Finally, retinal changes have been observed in amyloid degeneration with symptoms usually of chronic parenchymatous nephritis. The diagnosis is made from the etiology (chronic suppurations), enlarged liver and spleen. In intermittent albuminuria, retinal hemorrhages have been seen.

Albuminuria occurs in pregnancy in 1 to 2 per cent. (Leber) and the retinitis in 1 out of 3000 cases. Severe cases of **nephritic retinitis** occur quite frequently in the **nephritis** of **pregnancy,** and are sometimes accompanied by uræmia or by eclampsia. The cause of the eye changes is always a nephritis which may be transitory; it is not the expression of a toxæmia which affects both kidney and retina similarly; there is an associated nitrogen retention. Its dependence on pregnancy is shown by its frequent appearance in women who have previously been healthy, by its rapid recovery on the termination of the pregnancy, its relapses in subsequent pregnancies and its comparatively benign course. The probability is that the kidneys are irritated by products of metabolism of the fetus. It is not likely that these same substances cause retinitis directly but that they must act through the intermediary of the kidney, especially as they seem to occur not in the beginning of the nephritis, but in the stage when a kidney insufficiency has developed.

The nephritis generally occurs in the first pregnancy and only occasionally in a later one without symptoms in preceding pregnancies. The retinitis appears usually in the last four months of pregnancy. The ocular changes occur in the retina (fibrinous exudations, hemorrhages, detachment) or at the papilla, and acute amaurosis may result during an eclamptic seizure. The retinal detachment regularly recovers and the retinitis may leave no traces, but more frequently shows an optic atrophy with signs of tissue degeneration and pigmentation. The vision is generally somewhat impaired. Relapses in subsequent pregnancies frequently occur.

Uremic Amaurosis.—This is a sudden, bilateral complete loss of sight, with cerebral symptoms and convulsions. There is suppression of urine. The eye grounds are perfectly normal on ophthalmoscopic examination, unless the attack came on during an albuminuric retinitis. The attack lasts twelve to twenty-four hours and after two to three days vision has returned to the previous state. Uremic amaurosis occurs in acute nephritis after scarlet fever, then in contracted and chronic parenchymatous nephritis and in pregnancy or labor.

The **ophthalmoscopic changes** can be classified roughly into three groups: I. So-called albuminuric retinitis. II. Hemorrhagic retinitis. III. Papillitis and optic neuritis. According to Schlesinger[1] the frequency of these groups is 77 per cent., 14 per cent., and 7 per cent.

I. ALBUMINURIC RETINITIS.—Leber divides the ophthalmoscopic changes of the first and most frequent group into three stages: (1) hyperemia and inflammatory infiltration; (2) fatty degeneration; (3) retrogression and atrophy.

First Stage.—The condition begins with a marked venous hyperemia in which the disc is red like the surrounding tissues. The veins are distended and tortuous, the arteries narrow and difficult to see; they sometimes show white lines. The surrounding tissue of the retina is hazy, especially along the greater blood vessels, and the eye ground assumes a dull grayish-red color. There may be a few hemorrhages, which later increase in number. They are situated in the nerve-fibre layer and are usually flame-like or linear. White spots appear early. At first they occur in conjunction with the hemorrhages and then they become very much larger and develop in places where there are no hemorrhages. These white areas are of two kinds. They are due to fatty infiltration and to ganglioform swelling of the nerve fibres. Occasionally the inflammation seems to be localized to the disc, which shows a decided swelling with a sharp drop toward the retina. The hemorrhages and white areas are then apt to be placed near the margin of the disc. Other changes may be absent and the picture will then resemble the choked disc seen in intracranial tumors.

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[1] Quoted from Groenouw, p. 99, *l.c.*

Second Stage.—The hyperemia continues and the changes in the tissue are those principally of fatty degeneration. The white areas enlarge and form a zone surrounding the disc and the macula lutea. The grayish opacity which surrounds the margin of the disc becomes more marked and may become white, with distinct radial striæ. About this clouded area there are large round or irregular-shaped glistening white or yellowish opaque areas of fatty degeneration which are in turn surrounded by zones of small spots of the same character. In certain cases these areas seem to coalesce and with the diffuse opacity surrounding the disc form an extended white surface ("snow bank").

When this stage is fully developed the hemorrhages are very small or are absent. In the region of the macula lutea, which is usually free from these large areas, there are small white spots of a glistening character like spattered white paint. The spots are arranged in rows which converge and represent a star-like figure. These spots depend upon the presence of fat globule cells in the external retinal layers and occasionally the vessels can be seen to pass over them. The location of these changes in the external fibre layer of the retina was demonstrated by Dimmer and Nuel. There are cases where hyperemia and circumpapillary clouding are poorly developed and scattered over the retina we find numerous punctate white spots ("degenerative form"). The macula may present a star figure. Hemorrhages are small and infrequent.

In some cases the white spots do not develop and the condition is that of simple hemorrhagic retinitis. In this form which most frequently develops in one eye only the case is not a true nephritis but the condition depends upon a presumable thrombosis of the central retinal vein. The vessels generally do not show pronounced vascular changes. In some cases a retinal detachment takes place. The symptoms of this detachment are those of an ordinary detachment with the addition of the retinal changes observed in nephritis. It is not infrequent to find ophthalmoscopic changes in the pigment epithelium, particularly in the periphery of the eye ground, and there may be small pigment dots in the macula.

Third Stage.—The retinal changes may clear up if the fundamental disease is recovered from or comes to a standstill. In other cases they continue indefinitely or their clearing up is imperfect and relapses are possible. In the favorable cases the most marked changes slowly disappear. Of the white areas the star-shaped punctate group at the macula exists the longest and remains after all the other tissues have again become normal. The disc becomes discolored, its margins are sharply defined, the arteries narrow with white lines. The changes in the pigment epithelium are of great diagnostic importance, as they show that the discoloration of the disc depends upon a healed retinal condition. In milder cases the vision becomes normal and the changes in the eye ground disappear without leaving any trace.

II. THE SO-CALLED HEMORRHAGIC VARIETY OF NEPHRITIC RETINITIS.—Though the areas of fatty degeneration preponderate over the hemorrhages in the usual form of nephritic retinitis, there are cases in which the hemorrhages both in number and size preponderate, and this condition persists throughout the entire length of the disease. If areas of fatty degeneration are added to the hemorrhages, their number and size is always a relatively limited one. The star-shaped figure in the macula is sometimes present. The condition corresponds to the hemorrhagic retinitis following thrombosis of the central vein.

Magnus, in 1873, separated this form of apoplectic albuminuric retinitis from the typical one, which he called retinitis degenerativa. Leber believes that it is important to separate this condition from nephritic retinitis, for though it may be associated with nephritis, its cause is probably circulatory, as this picture, hemorrhagic retinitis, occurs more frequently in diseases of the circulatory apparatus without albuminuria or without other apparent disease. This circulatory change is that of a thrombosis of the central vein. According to Leber, albuminuria is present in quite a large percentage (20 to 30 per cent.) of the cases of thrombosis of the central retinal vein. The nephritis is not pronounced, but a mild transient form of albuminuria in which the patients are generally well and have no other symptoms. Leber believes that these cases correspond to those described as a hemorrhagic form of nephritic retinitis. A characteristic, aside from the ophthalmoscopic condition, is the sudden onset with a marked loss of vision, which usually affects only one eye and generally terminates in secondary glaucoma.

Elschnig[1] has described, under the term "light atypical retinitis," cases which present but a few hemorrhages and several small areas of degeneration without hyperemia or œdema. Pick[2] believes these changes to resemble those found in septic conditions and in anæmia. There is also a degenerative form[1] in which there is no hyperemia and but very few hemorrhages, but numerous small white areas throughout the background.

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[1] Elschnig, Centralbl. f. pr. Aug., 1894, p. 350.

[2] Pick, Kl. M. f. Augenh'lk., 1901, Vol. XXXIX.

III. PAPILLITIS AND OPTIC NEURITIS.—In nephritic retinitis the papilla may be swollen and infiltrated and there are hemorrhages and degenerative areas closely situated about the disc, while the retina at a short distance away remains free or the pronounced picture of choked disc without involvement of the surrounding retina may be present. The ophthalmoscopic picture thus resembles that found in intracranial tumors and the clinical picture is still more confused if cerebral symptoms due to uremia are present. Cushing and Bordley,[2] believe that the swelling of the papilla in these cases is due to increased brain pressure following an exudation into the ventricles and into the subarachnoid space. The increased intracranial pressure has been demonstrated by lumbar puncture and by trephining. The papillitis is apt to develop gradually, without producing visual disturbances until suddenly a uremic attack with complete blindness develops. This attack differs from the usual uremic attack in there being an ophthalmoscopic change. The attacks are sometimes of a milder variety and repeat themselves. Sometimes this form of papillitis is complicated with other cerebral symptoms which simulate a brain tumor.

Leber mentions the occurrence in nephritis of an inflammation of the optic nerve, with moderate papillitis but without cerebral symptoms. This occurs independently or with the nephritic retinitis and presents characteristic functional disturbances in the form of a central scotoma or a ring scotoma.

Complications.—There may be transient obscurations or complete blindness, which are due to uremic attacks. Other complications are thrombosis of the central vein or of the central artery. An important complication is

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[1] Oglesby, B. M. J., 1877.

[2] Cushing and Bordley, J. A. M. A., 1909.

detachment of the retina. In a small number of cases the subretinal exudation which is often present to a slight degree increases and produces a distinct detachment of the retina. This occurs principally when the nephritis develops acutely and when the eye complication is an unusually severe one. Up to the present day 59 cases have been carefully reported with an anatomic examination in many of them. In 60 cases which Leber has collected, 25 occurred in the nephritis of pregnancy, and 35 in other forms of nephritis. The detachment of the retina is usually recognized by the same ophthalmoscopic changes as in other cases. It is sometimes stated that the bulging part of the retina does not waver. No retinal tear can be made out which, even in the other cases is usually not present. The ocular tension is not affected. Shallow retinal detachment often evades ophthalmoscopic recognition. The detachment affects nearly always both eyes. It differs from the ordinary form of retinal detachment which apparently occurs from a retraction process from within, by being caused by a primary secretion of fluid probably derived from the choroid (Leber). As to the outcome, there is a very remarkable difference between the detachment which occurs in the nephritis of pregnancy and in other forms of nephritis. In the former if the nephritis disappears on birth of the child, the retinal changes may rapidly improve and the detachment usually becomes reattached within fourteen days after labor (Leber), while in the ordinary detachment of the retina spontaneous recovery is a most unusual termination. The vision after reposition of the retina is, however, not always improved to a corresponding degree. There are quite a number of cases in which fair vision returned, while in others a diminution and even blindness resulted, notwithstanding disappearance of the detachment. The favorable outcome depends on the length of time the detachment has existed—in other words, from its early or late appearance during the pregnancy and the length of time which intervenes before labor. The unfavorable cases are those in which the nephritis existed before pregnancy and became aggravated through the latter. The cases of retinal detachment in nephritis which do not depend on pregnancy are very unfavorable. This complication is usually a sign of advanced disturbance of metabolism and heralds the early approach of death. Death usually follows with a rapid increase in the kidney symptoms, in a space of a few days to a few weeks or months after the onset of the detachment. There are, however, a few individual cases reported where detachment disappeared and life was prolonged for some time. This suggests a cautious prognosis and shows that a retinal detachment is not necessarily hopeless. Owing to the condition being a chronic one, a prolongation of life beyond a short time is, however, not to be expected.

Frequency.—The proportion in which retinitis occurs in nephritis is 22.4 per cent. (Groenouw), 32 per cent. (Leber). Corresponding to the occurrence of nephritis in general, the retinitis occurs at all periods of life. It is unusual under twelve; most cases occur between thirty to sixty years of age. Nettleship[1] states that in patients under twenty-one, retinitis or multiple retinal hemorrhages were found in 31 out of 80 cases of interstitial nephritis, in 2 out of 3 cases of interstitial nephritis associated with calculus, in 7 out of 149 cases of parenchymatous nephritis. In these 40 the total number of severe cases was greater than in adults. The earliest age was five. More than half were between fourteen and twenty-one years of age. Twenty-four of these 40 died at a known interval. This shows that the prognostic importance of the retinal changes is equally if not more grave than in adults. Interstitial nephritis in the young is much more frequent in females than males, while in parenchymatous nephritis the males are slightly in excess. Congenital syphilis is often the cause of acute infantile nephritis. The cause of the nephritis was one of the specific fevers including syphilis in 31 out of 45.

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[1] Nettleship, On Renal Retinitis in Young Subjects, R. L. O. H., XVI.

The disease in most cases is bilateral; occasionally it occurs in one eye, and the second eye is then involved after a certain length of time. The degree in the two eyes is not necessarily the same. A permanent affection of only one eye occurs, and is not so unusual as was formerly supposed; and there are cases on record where a pronounced retinitis was observed in one eye, in severe nephritis, where the other eye remains free from two and one-half months to one year up to the time of death. A case has been reported by van Milligen,[1] where the second eye remained intact for eight years.

The **course,** like in most chronic nephritis cases, is a gradual one. Vision is gradually diminished and then is apt to remain stationary for a long time. The visual disturbances are generally slight, often out of all proportion to the ophthalmoscopic picture; in general they undergo the same variations as the nephritis. If the kidney disease is recovered from, the retinal changes disappear, leaving useful and sometimes normal vision. Marked changes and even detachment may be recovered from. In the chronic cases in which complete recovery occurs but rarely, we may find that if the nephritis is improved the retinal œdema and the other changes gradually disappear. The last to go is the star-figure in the macula. In the most favorable cases certain changes remain definitely present. If the condition lasts through a number of years, changes in the vascular walls are usually added and the disc will show a whitish discoloration and the picture of retinal atrophy develops. Transient blindness is due to an intercurrent uremic attack. The majority of the patients, however, die from the primary disease before any changes occur in the retinal picture.

The retinitis is a direct result of the kidney lesion, where certain injurious agents coming from the blood act upon the vessels and the tissues of the retina, and generally indicate that the kidney disease has reached a certain degree of severity and will briefly lead to death. Miles has tabulated 105 cases with acute and chronic nephritis. Of these, 53 per cent. with albuminuric retinitis died, while 27 per cent. without an eye complication died. Numerous statistics have shown the unfavorable prognosis of albuminuric retinitis, and according to most of these, death results from one to two years, in some after two to three years. In Geis,[1] series of 38 cases, 29 died within one year, 4 within one to two years, and 2 within two to four years. According to most observers, the prognosis is death in 90 per cent. within two years.

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[1] Quoted from Leber.

Isolated retinal hemorrhages in nephritis are usually due to the blood-pressure increase and to the cardiac hypertrophy, and consequently only appear in the chronic interstitial nephritis. The isolated hemorrhages occur from the rupture of the diseased vessel following increased pressure. In judging of the severity of albuminuric retinitis, cases of isolated hemorrhages in retinitis should be considered apart. While albuminuric retinitis is a sign of the terminal stage of the kidney process, retinal hemorrhages point rather more to increased arterial pressure acting upon diseased vessels, and in these cases apoplexies of the brain are to be expected.

In Geis' cases with isolated retinal hemorrhages, hemorrhages in the brain always occurred. While patients with albuminuric retinitis, apoplexy occurred in only 9 (one-third to one-fourth), about as many died from uremia. Thrombosis of the veins occurring in cases with albumen in the urine should be sharply differentiated from cases of albuminuric retinitis and isolated retinal hemorrhages. These thromboses, as has been stated, may occur from comparatively slight local anatomic changes which rob the condition of its serious prognosis.

The course of the **albuminuric retinitis** of **pregnancy** is only influenced by the labor and the final prognosis depends on the nephritis. Diet can do nothing. This is always a serious condition and even if the process is arrested by the induction of labor, the vision suffers permanent damage. Prophylactic treatment is treatment of the eclampsia (careful examination of the kidney function, blood pressure and nitrogen ratio in the blood; diet, rest in bed, venesection). When the retinitis is present, labor should be immediately induced. This always relieves the retinal condition. If the retinitis appears in the first six months, the pregnancy should be immediately arrested, in later appearing retinitis, during the last seven weeks, it is permissible to wait when the retinal changes have not advanced too far. Subsequent pregnancies should be avoided unless the kidneys become normal for two years under careful watch of the urine. In many ways the retinitis of pregnancy differs from the usual albuminuric retinitis. The retinitis, just as the kidney in pregnancy, may recover more or less completely, and the condition may relapse in later pregnancies. According to Culbertson, 16 per cent. recovered completely, in 58 per cent. there was a partial recovery of vision, while 24 per cent. remained blind. The prognosis as to life is comparatively good. The persistence of albuminuria for years after pregnancy does not necessarily modify the prognosis.

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[1] Geis, Kl. M. f. Augenh'lk., XLIX, No. 1, 1911.

Pathology.—A definite explanation of the connection between the nephritis and retinitis can not be given. There are apparently two factors present—the disease of the blood vessels and the altered condition of the blood; sclerosed vessels may be secondary to the latter condition or a part of a general angiosclerosis. Accordingly, two forms of albuminuric retinitis can be distinguished: I. When the altered blood condition is responsible, causing the acute nephritis, as after scarlet fever and pregnancy. II. Where the general arteriosclerosis causes the chronic nephritis.

The changes in the vessels which occur in these cases were particularly studied by Michel and Karl Theodor, and all changes were made dependent upon this cause, so that Michel believed that the retinitis did not follow nephritis but that both were produced by a common cause, resting in changes in the vessels. Leber believes that the changes in the small retinal vessels in nephritic retinitis can no longer be regarded as the cause, as these same changes in the retina have been observed with normal vessels, and the vascular changes are different, as the retinal affection is acute or chronic.

Elschnig examined about 200 cases of nephritis. In these a nephritic retinitis of various grades was found present in 32 per cent. of the cases. In the remaining 67 per cent. in more than one-half the vessels were normal, and slight arteriosclerosis was present in 44. The changes were in general of very slight degree. There never was marked or extensive narrowing or clouding of the arteries. And in the cases of albuminuric retinitis in only a small part where the process was particularly severe were marked vascular changes present. The direct influence of nephritis in the causation of changes in the retinal vessels is probably very slight. The changes in the retinal vessels found in cases where a nephritic retinitis has not developed are probably not the result of the nephritis, but an associated general arteriosclerosis.

Opin and Rochon-Duvigneaud are also opposed to this vascular theory and Shieck showed the inconstancy of the vessel changes. Rochon-Duvigneaud[1] is sharply opposed to the view that a common cause affects the vessels in both kidney and retina equally. Opin[2] and his investigations have shown that in nephritis no organs are affected except the kidney, retina and hypertrophied heart, and that there is no generalized vascular affection. The distinction between a retinitis secondary to a primary nephritis with changes in the blood (Samelsohn and Weeks) and a retinitis from general vascular disease is too arbitrary in his opinion.

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[1] Rochon-Duvigneaud, Soc. franc. d'Ophth., 1912.

[2] Opin, Journal de Physiol et de Pathol. gen. IV, No. 6, 1903.

Rochon-Duvigneaud believes that there is a NaCl and urea retention in nephritis and a pathologic increase of urea in the blood. Morax and Weill[1] have shown that in many cases there is a parallelism between the retinal changes and nitrogen retention (azotemia). Onfray and Balavoin found a hypoviscosity of the blood and raised blood pressure.

Moore[2] suggests that there are two factors responsible for the retinal changes in renal retinitis, the one vascular and the other toxic, of which either gives characteristic changes. Thus in the chronic parenchymatous nephritis the toxic element is much more prominent than the vascular one. The blood pressure is not raised and the retinal changes consist in many areas of white exudation in the tissues of the retina, often accompanied with œdema of the retina which may be so great as to lead to detachment. Retinal hemorrhages are not plentiful and the usual star figure is not characteristic. In the other variety, chronic interstitial nephritis, which is associated with high blood pressure, the retinal vessels show sclerosis, hemorrhages are characteristic and the star figure in the macula is generally present. There are usually but few exudates. Of course, both factors may enter into the causation of a particular case.

Other Eye Lesions in Nephritis.—ŒDEMA OF THE EYE LIDS is one of the well-recognized symptoms of nephritis. The œdema rarely affects the ocular conjunctiva. It generally remains for a few hours or days and then disappears for a time. Every non-inflammatory œdema of the lids does not indicate a renal or cardiac lesion, as this condition occurs apparently in perfect health. **Subconjunctival hemorrhages** have been observed in nephritis usually in association with retinitis albuminurica. They occur so readily from various causes that they are of little significance.

Iritis and **irido-cyclitis** are very unusual complications.

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[1] Morax and Weill, Ann. d'ocul., CXLIII, p. 354, 1910.

[2] Moore, Quarterly Journal Medicine, 1916–17.

In Gutmann's statistics (see p. 396), chronic nephritis is put down as the cause for iritis in 5 per cent., while in most text-books this connection is not even mentioned. The writer has observed one case of cyclitis and a case of deep keratitis which occurred in patients with chronic nephritis, in whom no other etiology could be found.

A number of attempts have been made to associate **cataract** with albuminuria, and notwithstanding many statistics which have shown the frequency of albuminuria in cataract patients, it is now generally accepted that nephritis has nothing to do with the development of cataract. Groenouw states that cataracts do not occur more frequently in nephritis than in the healthy.

Patients with chronic nephritis whose general health has suffered, sometimes complain of a **weakness of accommodation.**

Ocular muscle paralyses have been described in the course of nephritis. These were due to cerebral hemorrhages,[1] which are usually recovered from but are of ominous significance. Hemorrhages and retinitis have been described in intermittent albuminuria (Ostwalt[2]).

In nephritis there is sometimes a general hemorrhagic tendency and this may affect the eye in the form of numerous retinal hemorrhages, of hemorrhages in the choroid, of the iris, conjunctiva, eyelids, Tenon's capsule, orbital tissue, and of the optic-nerve sheath.

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[1] Dunn, Archives of Ophth., 1898.

[2] Ostwalt, Rev. gen. d'Ophth., 1898.

XI. DIABETES[1]

Diabetes means the excretion of grape sugar in the urine; the body can not use the starch (or the resulting grape sugar) and thus nutrition suffers; the unburnt-up sugar in the urine means loss in calories, and the inability to burn up carbohydrates means danger in the production of acetone bodies (acetone, aceto-acetic acid, β-oxybutyric acid). To correct the nutritional defect, albumen and fat can replace the carbohydrates; it is necessary to see how much carbohydrates the patient can take care of.

The frequency of eye complications in diabetes is uncertain; the figures in statistics vary from 20 to 33 per cent. It is usually a disease of later life, between fifty and seventy years. It does not necessarily involve a grave prognosis for life as albuminuric retinitis does, but optic-nerve lesions and degenerative retinal affections indicate a severe general disturbance. The affections of the eye, in order of frequency, according to Groenouw, are: cataract 30 per cent.; retinitis 23.5 per cent.; optic neuritis 5.7 per cent.; ocular-muscle disturbance 3.9 per cent.; then come refractive changes (including accommodation) vitreous opacities, iritis. thrombosis of retinal vein, hemorrhagic glaucoma, and optic atrophy. In most cases the diabetes has existed for from five to twenty years and the patient's general condition is fair.

According to Hirschberg,[2] the most frequent disturbance is a **contraction** of the **range** of **accommodation.** This occurred in one-third of Hirschberg's cases. The reading glass is no longer suited for the age of the patient. This disturbance of accommodation occurs in both the mild and the severe cases. It is difficult to decide whether it is due to general muscular weakness or a special disturbance of innervation. On treatment of the general condition the weakness of accommodation very rapidly disappears.

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[1] Groenouw, Graefe-Saemisch, II ed., 1904.
Leber, Graefe-Saemisch-Hess, II ed., 1915.

[2] Hirschberg, D. m. W., 1887, Nos. 17–19.

Changes in refraction are common. The hypermetropia may increase, though this is probably the result of the changing of latent to manifest hypermetropia. The onset of myopia is often observed. When this occurs late in life and without lenticular change, diabetes must be suspected. This myopia may exist for some time or disappear as the sugar diminishes. It is probably due to increase of the refractive index of the lens as is frequently observed in incipient cataract, and not to a change in the aqueous.

The **iritis** is not characterized by any peculiarity in its clinical course. The symptoms are not very severe, though hypopyon and a fibrinous exudate may be present. The course is favorable, though complications may ensue. Microscopically the pigment epithelium on the posterior surface of the iris shows a remarkable change; the cells are hydropically swollen with scanty pigmentation. This change extends back to the ciliary epithelium. On occasion of an iridectomy, under these conditions, the aqueous turns black. This condition, in the writer's experience, has indicated a certain tendency to iritis in the after-course of a cataract operation.

Cataract.—The appearance is often not characteristic. In old people with diabetes the opacity begins about the nucleus, not directly under the capsule. In other cases, where the formation is bilateral and occurring in the young, the appearance is characteristic and striking, as was first pointed out by Foerster. There is a uniform subcapsular opacity, a bluish homogeneous sheen. The deeper parts are at first transparent, then they become opaque and the cataract now resembles the soft cataract of the young or the one consecutive to a retinal detachment. Diabetic cataract appears equally in both sexes and at all ages; the patients are often in good bodily health. The progress is usually rapid.

Hirschberg[1] says that diabetic cataract has the following importance: I. It can not be caused to disappear by the reduction of sugar in the urine. II. The prognosis of operation is nearly as good as that of the ordinary cataract. III. When diabetes has existed for over ten years there is regularly an opacity of the lens, even in middle age.

This cataractous formation has been brought into etiologic relation to sugar being found in the ocular fluids, but the quantity of sugar present is much too small. A more important change is the curious hydropic condition of the pigment epithelium just described. It is wise to improve the patient's general condition and reduce the percentage of sugar before operation. Even if it is still present, if the patient's general condition is good, the operation can be undertaken. Statistics of several operators, Uhthoff and others, state that their results equal those in the ordinary cataract. Elschnig does not hesitate to operate unless acetone and diacetic acid are present. The experiences of many operators show that the result of the operation is not necessarily worse than in the usual cases. In the writer's opinion there can be no question of the greater frequency of iritis and often severe irido-cyclitis as a complication after cataract extraction. This can be guarded against by a course of strict dieting, reducing the sugar in the urine. The presence of sugar is not a contraindication if the general bodily health is good, which means that there is no acetone or diacetic acid present. It is advisable to keep the patient in bed as little as possible.

The **retina** in diabetes, according to Leber,[2] is affected in a number of ways, particularly on account of a complicating nephritis; there are cases which are entirely independent of the nephritis, others depend upon the nephritis produced by the diabetes, and in still others the original picture of diabetic retinitis is modified by the addition of nephritis.

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[1] Hirschberg, D. m. W., 1891.

[2] Leber, Graefe-Saemisch-Hess, II ed., 1916.

There are forms of retinitis in diabetes in which the ophthalmoscopic condition is characteristic. This diabetic retinitis, though it resembles the nephritic retinitis differs from it as follows, as has been pointed out by Hirschberg:[1] (1) the disc is normal and there is no hyperemia or œdematous clouding of the surrounding retina; (2) there are groups of small sharply defined white spots, sometimes of angular form, which are situated principally in the region surrounding the optic nerve, between the superior and inferior temporal blood vessels. The changes may extend to the nasal side. The white areas may have notched margins or appear as tortuous streaks or half rings or irregular figures, without ever assuming the radiating star figure seen in nephritis.

Throughout the region of these areas and extending into the periphery there are many small punctate or striated hemorrhages. The condition never leads to the formation of pigment. The bright spots may undergo variations but do not disappear, but slowly increase in number and in size. Retinal hemorrhages are not uncommon without hyperemia or blurring of the disc margins, or white areas. The number and size of the hemorrhages varies. Sometimes there are only a few, sometimes there are groups of small blood spots, or they may be numerous and extended over the entire eye ground. Not infrequently they appear only in one eye. The hemorrhages may recur and break through into the vitreous, producing opacities. This dense hemorrhagic infiltration is followed by preretinal connective-tissue formation and the development of new blood vessels in the vitreous. Hemorrhagic glaucoma is not an infrequent outcome. The vessels themselves show no changes. These changes in the retina do not in any way show anything characteristic of a diabetic origin, as they may occur in a number of other conditions. Leber, however, believes that the hemorrhages occurring in diabetes, as compared with those in nephritis, show a tendency to break into the vitreous and produce recurring vitreous hemorrhages.

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[1] Hirschberg, D. m. W., 1890, Nos. 51 and 52.

Retinal hemorrhages in diabetes, Hirschberg,[1] divides into four groups: (1) small, punctate hemorrhages; (2) larger hemorrhages with vitreous opacities; (3) hemorrhagic infarct of the retina; (4) hemorrhagic glaucoma. A main characteristic in all uncomplicated cases is the absence of ophthalmoscopically visible changes in the vessel walls.

The amount of visual disturbance varies according to the seat of the pathologic process. A central scotoma is present when the condition is complicated with retrobulbar neuritis. In some cases an embolism of the central artery has been observed, generally before the onset of diabetic retinitis. As to the nature of the white spots in the retina, we only know of the ganglionic swelling of the nerve fibres. The cause for this diabetic condition in the retina is not known. In Uhthoff's statistics two-fifths of these retinal changes were monolateral. This is of importance from a differential diagnostic point. As is well known, many diabetics show symptoms of albuminuria. This does not mean that the condition is albuminuric retinitis, as the albumen is only an expression of the irritation of the renal epithelium.

True nephritis in diabetes is rare. It has been claimed by some that diabetes can cause the ophthalmoscopic picture of albuminuric retinitis; these cases, according to Kako,[2] must be extremely rare. In the later stages of diabetes a comparatively frequent complication is nephritis, which may change the retinal condition and the prognosis. Diabetic retinitis usually occurs in advanced years, between forty-five and sixty-five.

The course is a tedious one, because the changes occur in inveterate and confirmed cases of diabetes, where recoveries are very exceptional. The vision, however, may remain fairly good for many years. Nettleship[1] has shown that patients with this condition may continue their work for many years, though there are a number of cases on record in which the patient has lost his sight several years before death. Secondary glaucoma is apt to occur after a severe vitreous hemorrhage. Diabetic retinal changes belong to the late complications of diabetes, and it is generally found that the diabetes has existed for a long series of years. The patients not infrequently suffer from other complications of diabetes.

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[1] L. c.

[2] Kako, Kl. M. f. A., 1903.

The retinitis, as compared with the retrobulbar neuritis, Leber thinks, has a more serious prognosis, as the latter condition appears in the earlier stages. On the other hand, the patient's general condition need not have necessarily suffered when the retinal process begins, as many of these patients still consider themselves in good health, and it is not infrequent that the examination of the eyes first leads to a diagnosis of diabetes.

The condition occurs nearly exclusively in advanced age. Most statistics place it between the fifty-first and fifty-sixth year, though there are some cases which have occurred between the thirtieth and fortieth year. Diabetic retinitis, like nephritic retinitis, usually occurs in both eyes. If it is seen in only one, it will show itself in the presence of multiple retinal hemorrhages.

The prognosis is doubtful, surely much poorer than diabetic neuritis. While in this, with great probability, the condition will recover or improve, if the amount of sugar is reduced, the retinal lesions are much more obstinate, as it is very difficult to keep the amount of sugar down and the necessary diet can not be followed out. Even if the amount of sugar is reduced, the changes in the retina may not disappear promptly and often the vision does not return, as is to be expected. On the other hand, the prognostic importance as to life of diabetic retinitis is not as unfavorable as that of nephritic type. The cause of death is generally diabetic coma; many suffer from apoplexies.

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[1] Nettleship, R. L. O. H., 1904.

Geis[1] found in a study of 16 cases of diabetic retinitis that apoplexies occurred in about one-half of the cases. He does not regard the vital prognosis as necessarily unfavorable, although diabetic retinitis, like other diabetic diseases of the eye, generally occurs after the diabetes has existed for quite a long time and the body has been weakened. Hemorrhages occur in milder cases of diabetes, and definite prognosis can not be made from their occurrence; they frequently depend upon damage to the vessel wall, and aside from the isolated diabetic retinal hemorrhages, the blood pressure was normal in many cases.

Of Geis' 13 cases, 5 died within one to two years; 3 after four, five and six years; 3 were still alive after two and three years; and 2 after five years. A definite prognosis in diabetes, according to this author, seems only to be furnished by the isolated retinal hemorrhages, as these are frequently the precursors of hemorrhages of the brain.

According to Heine, in about one-half of all patients with diabetic eye changes, especially with retinal changes, death results within two or three years.

Nettleship[2] says that the presence of diabetic retinal changes does not point to the probability of early death as it does in chronic Bright's disease. In 48 patients, 38 are known to have died; no less than 28, nearly two-thirds or 60 per cent., lived more than two years; and only 9, or one-fifth, died within one year. In his nephritis cases (pregnancy excluded) one-third lived two years and two-thirds died in twelve months.

Diabetic retinitis is a more local affair than the retinitis in Bright's, for the changes in the retina progress even when the general condition is improving. Prognosis is therefore to be guarded for visual improvement. As for the complicating nephritis, the retinitis is always due to the diabetes; of the patients with this complication one-half to two-thirds died directly from their diabetes.

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[1] L. c.

[2] L. c.

The optic nerve is affected in the form of a **chronic retrobulbar neuritis.** There is a relative central scotoma with or without temporal pallor of the optic disc. This complication, according to some authorities, is always the result of protracted diabetes where the general health has suffered greatly, and the prognosis is necessarily bad. Groenouw is more optimistic and believes the prognosis is not bad and that in many cases with proper treatment improvement and recovery occurs. The course is, however, slow.

The scotoma frequently exists for blue or is absolute in contradistinction to the condition usually found in toxic amblyopia (Uhthoff). This condition can improve or remains stationary. Naturally there are mixed forms where tobacco and alcohol have played their part, though there are undoubted cases where this toxic influence does not enter. The age is also different, the neuritis in diabetes occurring much later (fifty to seventy years) than in toxic cases (thirty to fifty years). Simple progressive optic atrophy does not belong to diabetes.

Ocular paralyses are not infrequent in diabetes and the symptom, diplopia, should always suggest an examination of the urine. The VI nerve is most frequently involved, then the III and combined forms. The prognosis is good and recovery ensues after appropriate treatment unless the case is one with cerebral symptoms.

Hoffmann[1] has examined anatomically the eyes of three diabetics and found extensive disease of the nerve fibres, not only of the axis cylinders but also of the medulla, characterized by the appearance of glycogen.

Lipemia retinalis[2] is a peculiar ophthalmoscopic condition in which the retinal vessels appear as if they were filled with milk, though the real cause is an opacity of the plasma which occurs when the quantity of fat in the blood is raised beyond a certain percentage. The condition has usually been observed in young diabetics who are bordering on coma and suggests a grave prognosis, though recovery may take place.

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[1] Hoffmann, A. f. A., Vol. LXXIII, p. 261.

[2] Moore, Lancet, Feb. 20, 1915.
Koellner, Z. f. Augenh'lk., Vol. XXVII.
Darling, Arch. of Ophth., Vol. XLI, 1912.

In **diabetes insipidus,** a condition characterized by excessive urine and thirst without sugar, a number of ocular disturbances have been recorded. These are usually affections of the optic nerves or tracts or of the ocular muscles which are more directly connected with the cerebral disease than with the associated diabetes.

XII. DISEASES OF THE FEMALE GENERATIVE ORGANS

Diseases of the female generative organs were formerly regarded as responsible for practically all forms of eye troubles which occur in women (Mooren). With the present advance in diagnosis of general diseases this relationship has been given up as too indefinite. It can, however, be conservatively maintained that menstruation and its disturbance may aggravate and exert an unfavorable influence on the course of a preëxisting eye trouble.

In normal **menstruation** many women develop blue rings and œdema about their eye lids; chemosis has also been observed. The effect on an existing eye disease can be observed by hyperemia, an increase of the inflammatory signs or hemorrhages. In the writer's experience this was shown very distinctly by the appearance of a hemorrhage at the margin of an active choroidal patch in a young woman at the menstrual period. Suppression of the menses, particularly, has been held accountable for a number of ocular disturbances, such as retinal and preretinal hemorrhages and acute retrobulbar optic neuritis. It seems doubtful whether etiological factors (nasal sinus disease, multiple neuritis) have not been overlooked in connection with the last-named disease, whose importance has been recognized only in recent years. Inflammatory conditions of the uterus just as any septic focus in the body, especially febrile conditions complicating labor, may produce iritis, iridocyclitis, metastatic ophthalmia (see p. 247), retinal hemorrhages (septic retinitis) and optic neuritis. Excessive uterine hemorrhages have been a frequent cause of loss of sight with optic atrophy, the so-called amaurosis from loss of blood (see p. 407). Displacements of the uterus may cause reflex ocular disturbances, both functional and neuralgic.

Important eye changes occur in pregnancy, the puerperium and lactation whose relation to these states is quite definite, and the condition may be severe enough to necessitate an interruption of the pregnancy.

Pregnancy.—The increase of pigmentation in the skin shows itself also in the eye lids. The development of **pulsating exophthalmos** in a large number of cases seems to have occurred during pregnancy. In Sattler's statistics of 32 idiopathic cases, 23 occurred in women where the onset coincided with pregnancy and labor in 7. After labor the symptoms were aggravated. The condition occurred in these women in their late pregnancies and it began suddenly. Intermittent exophthalmos, a condition which becomes noticeable on bending forward, depending on a dilatation of the orbital veins, began in a case of the writer's after labor.

That sight may be affected during pregnancy and labor, has been known for many years. These transient cases with normal eye grounds are uremic. Recently attention has been drawn to an impairment of vision and to an affection of the optic nerves during pregnancy, probably due to a condition of toxemia which is called the **toxemia of pregnancy** and in which a nephritis is absent or only slightly marked. Thus, transient blindness occurring in icterus gravidarum has been reported by Groenouw;[1] the eye grounds were normal and the urine was free from albumen. The optic nerve is affected in pregnant women under the picture of an optic neuritis. Thus, chronic retrobulbar neuritis has been observed with the presence of a central scotoma. Uhthoff found in 66 cases of chronic retrobulbar neuritis 4 women with advanced pregnancy. The loss of sight usually does not begin before the fourth month and often in the seventh to the tenth month. After labor there is an improvement in vision, though recovery is often only partial. In subsequent pregnancies the optic nerve and the field may be still further affected. The association of this

[1] Groenouw, Graefe-Saemisch, p. 183.

optic nerve condition and pregnancy is particularly shown by relapses which occur in later pregnancies. These attacks of blindness differ from the uremic, as their onset is slow and the urinary changes are slight. This neuritis is probably due to an autoinfection, just as a similar condition has been observed in the puerperium and during lactation.

The writer[1] reported on 3 cases of neuritic atrophy with no other change in the eye grounds occurring in pregnant women with manifestations of toxemia. Whether a central scotoma was present at the beginning, could not be determined as no case was seen in the early stage. Vision is frequently regained or it remains defective with peripheric contraction of the field and in most cases the eyes become permanently blind.

According to Stone[2] the evidences of toxemia of pregnancy are as follows: In the early months nausea and vomiting; skin manifestations (urticaria). These conditions are all observed in normal menstruation. During the later months there is headache, loss of sight, vomiting, vertigo, neuralgic pains, œdema. The urine may contain albumen and casts. This may be followed by convulsions and coma. Rarely patients will become totally blind, comatose and die.

It is now generally accepted that hyperemesis gravidarum and eclampsia are expressions of a toxic state in which the liver is the seat of the chief lesion, often presenting the same changes as acute yellow atrophy. During the period of lactation there may be a continuation of this state of the system, and these symptoms may consequently persist. One of the most frequent is the insanity.

Chemical examination of the urine by Ewing and Wolff[3] has shown that there is a disturbance in the nitrogen ratio. The total amount of nitrogen is decreased. The nitrogen which is converted into urea is decreased, while the other nitrogen derivatives are increased.

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[1] Knapp, Affections of the Optic Nerve During Pregnancy, T. A. O. S., 1907.

[2] Stone, W. S., Toxemia of Pregnancy, Amer. Gyn., December, 1903.

[3] American Journal of Obstetrics, April, 1907.

In the fatal cases post-mortem examination has shown parenchymatous multiple neuritis (Lindemann) and acute degeneration of the liver.

It is important to realize that this toxemia may affect the eye sight or even cause blindness without the intermediary of a nephritis, and the question of the interruption of pregnancy comes up for decision in this condition just as it does in the well-recognized albuminuric retinitis of pregnancy.

Stephenson[1] describes cases in which sight was completely lost after labor in a woman who had suffered from headache and albumen. She was semi-conscious. The fundus showed no changes. The second case showed typical eclampsia. He believes that these changes occurring during pregnancy or labor, although often associated with headache, eclampsia and albuminuria, are not proven to be dependent upon uremia, though they are generally classed as uremic amaurosis.

Woods[2] mentions as unusual complications of pregnancy, ocular disturbances in the form of hemianopsia or a loss of some part of the field without retinal lesion, though sometimes with pallor of disc and a neuro-retinitis with hemorrhages. Holden[3] observed in a pregnant woman with the symptoms of toxemia, a retrobulbar neuritis, œdema of the macula and paralysis of the external rectus; the retina subsequently developed some pigmentation. Vision was regained after the induction of premature labor.

Albuminuric retinitis of pregnancy, as is well known, is a frequent cause for visual disturbance in pregnancy and labor (see p. 416). Rochon-Duvigneaud[4] regards it as due to a nephritis which in turn is the result of the toxemia of pregnancy.

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[1] Stephenson, Ophthalmoscope, Vol. VIII, p. 168.

[2] Woods, J. A. M. A., 1909.

[3] Holden, J. A. M. A., 1914.

[4] Rochon-Duvigneaud, Soc. franç. d'ophthal., 1912.

Labor.—During labor transient blindness may occur which is generally uremic in character and is frequently associated with eclampsia. In other cases albumen is not present in the urine.

Puerperium.—The most important lesion during this stage is puerperal fever, a form of septico-pyemia from infection of the uterus. The eye complications are purulent metastatic choroiditis and septic retinitis (see pp. 247, 251). An inflammation of the optic nerve in the form of optic neuritis has been observed in this stage, just as in the preceding ones, and it is probably due to an auto-intoxication, as albumen is absent in these cases. The other evidences of toxemia which are suggestive in determining the cause of the eye condition are the pronounced headache, the continuous vomiting and skin lesions. Examination of the urine has shown a nitrogen retention.

The tendency to the formation of thrombi can give rise to emboli. If the vessels are occluded with septic material, metastatic ophthalmia results. A bland embolus does not necessarily lead to the loss of the eye, though sight is very much affected. Hemianopsia has also been observed. These cases have been associated with milk leg. In one case of the author's, in which recovery took place, there were 7 thrombi in different parts of the body and in both retinæ numerous hemorrhages were present.

Lactation.—Women who are apparently in good health during lactation may suffer from loss of eye sight. The ophthalmoscope reveals an optic neuritis. The condition continues for months, and slight atrophy results, though the vision is but little involved. It occurs not only in the first but also in later births. This symptom has been observed not only in cases where nursing has been unduly prolonged or where the secretion of milk has been suddenly arrested, but also in apparently perfectly normal lactation. Apparently the eighth week seems to be the time in which this condition most frequently appears. In one case which Groenouw mentions the child died directly after birth, the secretion of milk continued for seven weeks, and shortly after this the disturbance of vision presented itself. The condition has been observed in one or in both eyes and the symptoms of a retrobulbar neuritis were present. The degree to which the sight is affected varies. The onset may be sudden and may lead to complete blindness. Recovery occurs after a number of months. General symptoms, headache, malaise and chills have sometimes accompanied the onset of the loss of vision. In other cases other nerves beside the optic nerve were found affected. The outcome is a favorable one, inasmuch as permanent blindness has not been observed, though a certain amount of reduction of vision may be present. The cause seems to be, by exclusion, a form of auto-intoxication.

Birth Injuries.—It is not infrequent that the eye of a child suffers during labor from the application of forceps. These injuries may be external, causing subcutaneous hemorrhages, swelling of the lids, or injuries to the cornea. The form of the injury which the **cornea** suffers may be divided into two varieties. In one there is a more or less dense opacity of the cornea which appears directly after birth and generally disappears completely after a number of weeks. The opacity resembles that found in interstitial keratitis. The other form is one which has been particularly studied by Thompson and Buchanan,[1] in which a number of vertical lines of opacity develop in the cornea. These authors speak of faint, curved lines of "refraction opacity" on the posterior surface of the cornea running vertically. These are probably ruptures of Descemet's membrane and must result from a compression of the eye ball. They are not easy to see except with the aid of a loupe. Later an unusual degree of astigmatism develops.

The eye itself may be forced out of the orbit by pressure exerted by the forceps. The eye lids may be torn and hemorrhages may occur in the orbit which will cause exophthalmos. The bones of the skull are injured in a variety of ways and cause a corresponding change in the orbit. A vulnerable point is, of course, the external angular process of the frontal bone.

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[1] Thompson and Buchanan, T. O. S., 1903 and 1905, p. 295.

Paralyses of the ocular muscles through traumatism at birth are unusual. The facial nerve is the most frequently affected, though the paralysis is generally transient. Reese[1] has observed a paralysis of the sympathetic nerve in a newly born child with marked deformity of the skull. Panas[2] has drawn attention to the liability of the VI nerve to injury as it crosses the apex of the petrous pyramid in instrumental labor where the head has suffered lateral compression.

Hemorrhages in the retina are quite frequent in new-born children, even when the course of the labor has been uncomplicated. The hemorrhages seem to occur in normal labor, particularly in the first births, when the labor is protracted. A twisting of the cord is a contributing factor. In the first two days after birth examination of the eye grounds has shown the frequency[3] of hemorrhages to be between 21 and 32.6 per cent. They vary enormously in number and size and are usually situated about the posterior pole. They rapidly absorb and disappear after one week without leaving any traces. Some authors have described hemorrhages which occur exactly in the macular region which have led to a circumscribed detachment. It is assumed that in these cases permanent damage results which explains the condition known as congenital amblyopia. The cause for this is not definitely understood. Direct pressure on the eye by the forceps can be responsible only in certain cases, and then severe damage would be done to other parts of the eye. Thompson and Buchanan and Wolff have observed large hemorrhages in the retina and in the vitreous, as well as optic atrophy after instrumental labors.

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[1] Reese, N. Y. Eye & Ear Inf. Reports, 1896.

[2] Panas, Int. Ophth. Congress, Edinburgh, 1898.

[3] Koenigstein, Wiener med. Jahrbuch, 1881.
Schleich, Mitteil. a. d. Ophth. Klinik, Tuebingen, 1884.

XIII. DISEASES OF THE OSSEOUS SYSTEM[1]

Hyperostosis Craniæ (**Leontiasis ossia**) consists in an enormous thickening of the bones of the face and of the skull, occurring in young patients. The lower margin of the orbit is particularly affected. Exostoses may be present in both orbits and produce exophthalmus or papillœdema. The condition is general or localized. General hyperostosis is exceedingly rare. Narrowing of the optic foramen has caused optic neuritis and atrophy. The thickening of the bone may also cause a narrowing of the lacrymal canal with dacryocystitis. The disease is extremely chronic and is of unknown cause. In the diagnosis the following conditions must be considered: acromegaly in which enlargements of the hand and of the feet are present, myxœdema with characteristic changes in the subcutaneous tissue, osteitis deformans which usually attacks patients in advanced life and particularly the long bones. Cases of large bony growths encroaching upon the orbital contents have been operated upon. Green[2] has recently studied the ocular changes in leontiasis ossea and gives a synopsis of all reported cases.

Tower Skull (**Oxycephaly**).—This deformity is produced by the premature ossification of the coronary suture, leading to elongation of the skull backward and upward, while the forebrain remains rudimentary. There is a depression between the orbits in the region of the cribriform plate with shortening of the orbits whose upper walls are depressed and the large wings of the sphenoid are slanting. This leads to exophthalmus which is even more pronounced by the recession of the forehead. The pressure of the growing brain on the skull causes the impressiones digitatæ, which are apparent in the radiogram. The disturbance of vision begins in the first years of life (up to the fifth year) and does not progress after the seventh year. The eye changes are ophthalmoscopic and the optic nerve head presents the picture of post-neuritic atrophy, optic neuritis, choked disc, or simple atrophy.

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[1] Groenouw, Graefe-Saemisch Handbuch.
Uhthoff, Graefe-Saemisch Handbuch.

[2] John Green, Jr., Trans. A. A. Ophth. and Oto-Lar., 1915, p. 283.

In Uhthoff's statistics, post-neuritic optic atrophy was present in 65 per cent. When seen early the cases show distinct choked disc. This was observed in 18 per cent. and in patients before the seventh year. The cases do not go on to blindness and generally a fair amount of vision remains. Simple optic atrophy without signs of previous inflammation was observed in 10 per cent. Both eyes are affected but not necessarily to the same extent. Blindness occurs in 7 per cent. The visual field shows usually a concentric contraction. Other eye symptoms are divergent strabismus, nystagmus, and exophthalmus.

In the development of the eye condition, the growth of the brain in the deformed skull causes an increase of intracranial pressure which acts on the optic nerves like a brain tumor or hydrocephalus; after the growth of the brain has passed its active stage, no further injury to the optic nerve need follow. Some authors regard a serous meningitis as the active cause. Others claim a narrowing of the optic canal to be the essential feature. This Uhthoff regards as an exceptional condition and not proven. Another theory states that pressure is exerted by the dural prolongation of the optic canal.

The operative treatment depends on the accepted theory of the cause. An operation can only help if undertaken during the stage of the development of the visual disturbance, *i.e.*, when the optic neuritis is still active. The operation consists in lumbar puncture, trephining or callosal puncture; recently Schloffer has published an attempt to open up the bony optic canal.

Rhachitis.—Rhachitis is regarded by many to be one of the most frequent causes for zonular cataract. This form of cataract, characterized by an opaque zone surrounding a clear nucleus, is usually bilateral and is observed in the first years of life, though it may be congenital. These children frequently present other rhachitic manifestations, in the teeth, in the skull, in the bones of the extremities, and they is often a history of convulsions.

The deformities in the teeth consist in a defect in the enamel, by which it gradually diminishes from the root to the free surface and is often completely absent at the cutting edge. There also may be small holes in the enamel or distinct ridges or furrows. These changes are found in the incisors, the eye-teeth and the first molars.

Statistics on the frequency of rhachitic symptoms and zonular cataract are as follows: Groenouw mentions the statistics of von Arx[1] who found in 189 patients with zonular cataract, convulsions in 57 per cent., rhachitic changes in the teeth in 66 per cent., deformities of the skull in 32 per cent., changes in the bones of the extremities in 21 per cent. Baehr[2] found that in 153 cases of zonular cataract the condition was one-sided in only 7. Other symptoms of rickets were present in 79 to 89 per cent; changes in the teeth in 58 per cent., convulsions in 40 per cent. The convulsions usually are repeated and appear for months, generally in the first years of life. Symptoms of general rickets were present in 36 per cent. A number of explanations of the association of rickets and zonular cataract have been furnished. Horner believes that in the development of the normal lens a layer becomes altered on account of a rhachitic interference with nutrition, then when the cause is corrected, new and clear layers of lens matter are formed about the clouded zone. This process can be repeated a number of times, which explains the clouded layer appearing in two or three duplicates. The question of increase

[1] von Arx, Dissertation, Zurich, 1883.

[2] Baehr, D. m. W., 1900, No. 9.

in the case of a lamellar cataract is an important one and Spicer[1] says that if the nucleus is small, well-defined with a clearly cut margin and the periphery of the lens perfectly clear, no increase would occur. If small radiating lines of opacity extended from the edge of the nucleus toward the periphery, the opacity probably would progress(Critchett).

Bennett[2] believes that lamellar cataract is a disease of early infantile life, and that the probable cause is some general derangement of health which also affects the teeth (probably errors of feeding and nutrition). The dental enamel and the lens are embryologically similar and both affected by the same cause. Collins suggests that the part of the lens furthest from the source of nutrient supply should suffer in a disturbance of nutrition and contract.

Blue Sclerotics and Fragility of Bone.—The bluish discoloration of the eyeballs is due to a thinning of the sclera which is a congenital anomaly. A number of family trees have been examined and, in addition to this ocular anomaly, a pronounced tendency to fracture of the bones was found present. Burrows examined a family of 29 individuals in whom 13 had blue sclerotics and 9 suffered from fractures of the bones after slight causes. Stephenson found a family of whom 21 members in four generations were similarly affected. The common cause is regarded by Eddowes to be a congenital defect in the fibrous tissues in general.

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[1] Spicer, T. O. S., 1892.

[2] Bennett, T. O. S., 1901.

XIV. SKIN DISEASES

Eczema.—All extensive eczemas are associated with conjunctivitis, particularly when the eczema affects the skin of the face and of the head, as is so frequent in children. This conjunctivitis is a very serious condition. The conjunctiva is swollen and the discharge is considerable, though never purulent. The eye lids are swollen and excoriated; they are so inflamed that the small patient usually keeps them closed.

In addition to the conjunctivitis there is frequently an associated superficial keratitis. The discharge from the inflamed eye lids irritates the conjunctiva, whereby the catarrhal symptoms are increased, while the tears and secretion from the conjunctivitis in turn aggravate the eczema of the face. The children suffer from photophobia and keep their faces buried in the pillow. The retained discharge augments the conjunctivitis. The treatment of the conjunctivitis is of no avail until the eczema of the face is relieved.

The eczema causes frequently a secondary swelling of the cervical glands which quickly subsides on the proper treatment of the eczema. An important factor in the causation of the eczema are head lice. Phlyctenular disease of the conjunctiva and cornea is regarded by some to be akin to eczema and is often called conjunctivitis eczematosa. In every general eczema of adults a certain amount of conjunctivitis is always present. The conjunctivitis in these cases is the result of the same cause which produces the eczema of the skin and the conjunctivitis disappears upon the cure of the cutaneous lesion.

In some cases a conjunctivitis causes an inflammation of the skin of the eye lids, as after the use of atropin in certain predisposed individuals. This is regarded as an idiosyncrasy against atropin, though in the majority of the cases it is probably due to contaminated eye drops. Eczema of the lids usually heals without leaving changes. If it is neglected it may lead to loss of the eyelashes, thickening of the margin of the lids and ectropion.

Psoriasis is a parakeratosis characterized by small efflorescences which become covered with silvery white scales. On their removal a bleeding point is revealed. Psoriasis rarely affects the skin of the face and the eye lids; in the latter location it may cause the loss of the eye lashes and ectropion. Exceptionally psoriasis plaques have been found on the conjunctiva, though its occurrence in mucous membranes is questioned. There is a simple conjunctivitis which accompanies extensive cases of psoriasis and which can not be cured until the skin lesion disappears. Sometimes the conjunctivitis is due to an irritation from the remedy which is used for the psoriasis. Some authors have described patches of a red color in the conjunctiva in psoriasis and a patch may extend directly from the skin of the lid to the conjunctiva.

Rosacea.—Arlt first drew attention to the fact that rosacea of the skin of the face may produce complications in the eye. This author described a characteristic corneal change which consists in a shallow round defect in the corneal tissue, with a perfectly clean base. These ulcers result from the breaking down of small eruptions on the surface of the cornea. The conjunctiva is intact, though there may be ciliary congestion. The disease is unusually obstinate as these little ulcers only heal very slowly and are apt to relapse. Capauner[1] described five cases of the corneal complication and drew attention to their great importance. The beginning of the corneal complication, according to this author, consists in a curious vascularized nodule as large as the head of a pin, which regularly appears whenever there is an exacerbation of the rosacea of the face. The epithelium is desquamated and superficial defects develop. The corneal nodules are frequently surrounded by a uniform zone of opacity. As the infiltration gradually advances, extensive opacities of the cornea result. The lid margins sometimes show typical eruptions in the form of red nodules.

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[1] Capauner, Zeitschr. f. Augenhlk., Vol. XV, p. 126.

Erdmann[1] observed 21 cases and found that the disease affects women to a much greater extent than men. These patients furthermore are with few exceptions in the climacteric period; they usually are overnourished and full-blooded. Erdmann found that the eye disease appears in the warm periods of the year, especially in the spring, though there seems to be a connection between the severity of the eye complication and an active relapse of the skin affection.

The changes in the eyes consist in a seborrhœa of the lids, with secondary conjunctivitis which is the most frequent, and, as a rule, the only ocular complication of rosacea. Furthermore, red flat pimples are found on the margins of the lids, often covered with yellowish scales. The conjunctiva of the eye lids and the retrotarsal folds show the usual irritation, while characteristic changes are found in the bulbar conjunctiva. These consist in circumscribed or diffuse red areas, particularly in the palpebral fissure. In addition, there are small elevations on the sclera. These are usually situated near the limbus; they are not particularly painful and disappear after a number of weeks.

The corneal complications, according to Erdmann, consist generally in sub-epithelial grayish infiltrations which are slightly elevated, about the size of the head of a pin, with an opacity of the superficial layers of the cornea. In addition to these superficial infiltrations, deeper ones are observed of varying size, usually round and near the limbus. The surrounding parenchyma of the cornea is opaque. The infiltrations usually disappear after a few weeks. In the center there develops a chalky or a yellowish-white collection of necrotic tissue, which can be removed easily with a curette. Some of these areas of infiltration gradually extend in the form of a rodent ulcer and are accompanied with superficial blood vessels. They usually advance toward the center, so that finally the vision is very much disturbed. The corneal processes, both the superficial and the deep, are nearly always situated in the lower half of the cornea. Small chalky white sub-epithelial deposits are occasionally observed. It is noticeable that the interior of the eye is but little involved and iritis is a very unusual complication.

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[1] Erdmann, Archiv. f. Augenh'lk., Vol. LXVII, 1910.

The association of rosacea and pyorrhœa alveolaris was suggested by Hern, according to Lang (see p. 388). In one case of keratitis where this association was present and which was under the writer's care, no treatment of the corneal complication availed until the teeth were attended to.

Pemphigus.—This is a disease characterized by the formation of blebs in attacks which appear on the skin and the mucous membranes either in an acute or chronic form. It affects the skin of the lids and the conjunctiva.

It is difficult to define pemphigus exactly because, as Saemisch[1] says, opinions among dermatologists on pemphigus are not uniform and the condition which is regarded as pemphigus of the conjunctiva by ophthalmologists is not recognized by dermatologists as being a part of general pemphigus, and finally because the views of ophthalmologists still differ on the relation of pemphigus of the conjunctiva to the so-called "essential shrinkage."

Francke,[2] who has collected 107 cases in his monograph, suggests that pemphigus as it is a collective term should be dropped and the condition called "conjunctival shrinkage with bleb-formation" substituted.

The skin is affected in about three-fourths of the cases and the other mucous membranes in about one-half. The mucous membranes affected are usually those of the nose, throat, mouth and the larynx. Pergens[3] collected 112 cases of the pemphigus of the eye and found associated vesicles in the skin in 9 cases of acute and 68 cases of chronic pemphigus. In the latter, two-thirds of the cases also suffered from pemphigus of other mucous membranes. The formation of blebs was localized to the eye in 2. Essential shrinkage of the conjunctiva without any blebs was found in 16.

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[1] Saemisch, Graefe-Saemisch-Hess, Conjunctiva, p. 576, II ed.

[2] Francke, Der Pemphigus, Bergmann, Wiesbaden, 1900.

[3] Pergens, Pemphigus d. Auges, Berlin, 1901.

The eyes become affected at the same time that the skin does, though sometimes the ocular lesion may precede or follow after. Morris and Roberts collected 28 cases of pemphigus of the conjunctiva. The affection began in the skin in 16, on the mucous membrane in 4, in the eye in 8. The cases in which the pemphigus seems to be localized to the conjunctiva finds its analogon in those cases where only the mucous membrane and not the skin are affected.

The acute form is distinguished by febrile disturbances which continue for a number of days and during which blebs appear on the hands, feet, chest, back, genitals, mucous membranes of the mouth, pharynx and of the eye. Severe conjunctivitis and swelling of the lids have been observed. There was a general acute cutaneous eruption with blebs and a severe catarrhal conjunctivitis.

In the chronic form of pemphigus various changes in the conjunctiva of the eye have been observed. Sometimes the conjunctivitis is of a catarrhal or membranous type which recurs. Hemorrhages have been noted from the necrotic areas in the conjunctiva. Beginning with one of these forms of conjunctivitis, the characteristic shrinkage of the conjunctiva sets in, occasionally accompanied with slight irritation and the appearance of blebs. This shrinkage pursues an interrupted course, slowly leading to contraction of the conjunctival sac, and finally produces severe changes in the cornea. This shrinkage is often the only pathological symptom which the patient presents, though in many cases signs of pemphigus of the skin or of the mucous membranes have preceded the conjunctival condition or are simultaneously present. Saemisch states that pemphigus may develop in other parts of the body some time after its beginning in the eye.

In some cases the change in the conjunctiva is insidious, accompanied with hardly any symptoms of irritation until the slowly developing characteristic shrinkage is present. Blebs on the conjunctiva are not frequently seen, as they rupture easily. Their development shows itself by a rapid increase in the symptoms of irritation. The site of the blister will then be noted by a circumscribed red and uneven area covered with exudate. In some cases a bleb has been seen, varying in size, sometimes as large as a bean, situated in any part of the conjunctiva. According to Francke, the formation of blebs is present in about one-seventh of the cases. Michel states that the site of predilection for pemphigus in the conjunctiva is the inner angle of the lids and the lower half of the ocular conjunctiva. From this it extends to involve the upper fold and the palpebral conjunctiva. As the condition of shrinkage continues, the conjunctival sac becomes obliterated and a symblepharon develops. The cilia are displaced and come in contact with the cornea. They frequently grow from the inner margin or the intermarginal portion of the lid. As the cicatrization continues, leading to xerophthalmus, the margin of the lid is turned in and a union of the lid margins to one another, particularly at the inner angle, has been observed. Finally, there is a complete disappearance of the conjunctival sac. This occurs usually after a number of years. Becker[1] found in 56 cases of pemphigus slight shrinkage of the conjunctiva in 9.3 per cent., symblepharon in 19.4 per cent., complete adhesion of the eye lids in 5.35 per cent.

In some cases membranes and blebs do not appear and gradually without any symptoms of irritation the conjunctiva shrinks. This essential conjunctival shrinkage is possibly not always due to pemphigus (Groenouw). The disease always occurs in both eyes, though the involvement of the eyes may not be simultaneous; and it is therefore possible to observe the process in various stages in the same patient.

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[1] Becker, Inaug. Dissert., Jena, 1896.

The cornea is involved at various stages of the process, and in a variety of ways. Blebs, ulcers and infiltrations have been described; sometimes the cornea is covered with a vascular membrane like a pterygium, and in most cases the final stage is the transformation to a dry, horny skin.

Erythema Multiforme.—This skin lesion usually begins on the back of the hand or of the feet as well as the adjoining extremities in the form of red spots which rapidly enlarge. As it extends, the mucous membranes become involved. The patient often complains of general subjective symptoms and after from two to six weeks recovery takes place.

The conjunctiva has been affected in quite a number of cases. It has been particularly well described by Duering who found that in an epidemic in Constantinople conjunctivitis was frequently present. The ocular lesion often occurred at about the onset of the cutaneous eruption. In the region of the palpebral fissure there is usually a triangular area of conjunctiva with its base toward the margin of the cornea which is congested and presents a number of small elevations as large as the head of a pin. The rest of the conjunctiva is hyperemic and there is moderate discharge. The disease gets well in from two to three weeks. Michel has described an interstitial keratitis and marginal infiltrations of the cornea have been observed.

Herpes Iris.—This variation of the preceding disease is characterized by a crown of vesicles which appear at the margin of an erythematous patch. The conjunctiva, if involved, presents either a simple or a membranous conjunctivitis. The palpebral conjunctiva is red and hypertrophied, the ocular conjunctiva is congested and œdematous and there is some discharge. It differs from the ordinary conjunctivitis[1] by the absence of photophobia, pain and discharge, while the conjunctival œdema which is present is very striking. The severe form occurs as a membranous conjunctivitis and sometimes produces corneal ulcers.[1] The membranous type usually recovers. If the cornea and iris are involved, the prognosis is, of course, grave.

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[1] Hanke, Graefe's Archiv., Vol. LII.

Xeroderma Pigmentosum.—This unusual skin disease frequently affects a number of children in the same family. The condition develops in the first or in the second year of life in the form of circumscribed red spots on the skin. These only occur in that part of the skin which is exposed to the sun's rays. They disappear and reappear after renewed exposure to the sunlight. If this has continued for a number of times, freckles develop with pigment atrophy in the intervening skin, forming white islets. Later the skin shows a vascular dilatation of the capillaries. After some years the skin atrophies and contracts, causing ectropion of the lids. The skin then becomes peculiarly susceptible to the formation of cancer which finally leads to the death of the patient.

In xeroderma the eye lids and the conjunctiva are affected. The skin of the lid goes through the stages which have just been described, the eye lashes are lost and ectropion develops. Wart-like elevations appear on the skin which eventually turn into carcinoma. The conjunctiva just as other mucous membranes is sometimes affected. The symptoms are those of a conjunctivitis with circumscribed red spots, pigment deposits and teleangiectatic areas in the conjunctiva. The conjunctiva subsequently shrinks. Graefe has observed tumors of the conjunctiva in the late stages.

Favus.—Favus sometimes affects the eye lids without producing any disturbance in the rest of the body. It appears as a more or less extensive yellowish scab which includes the eye lashes but does not affect the margin of the eye lid. The scab presents the characteristic sulphur-yellow color and after its removal a bleeding red area remains. The scab shows on microscopic examination the characteristic spores and mycelium.

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[1] H. Barkan, Arch. of Ophth., Vol. XLII, p. 236, 1913.

Herpes Tonsurans.—The lids and the cilia are affected under the picture of a blepharitis. The characteristic is that the cilia are broken off close to the skin like stumps and are covered with scabs and scales. Upon examining the cilia the gonidia of the trichophyton are found.

Molluscum contagiosum occurs in the eyelids as one of the sites of predilection. There are small circular tumors of a whitish color with a central depression out of which a jelly-like material can be expressed. In certain obstinate conjunctival catarrhs these mollusca have been found at the margin of the lids and the conjunctivitis did not heal until the little tumors were removed (Groenouw).

Lichen ruber planus consists in small, brownish-red nodules which appear isolated or in groups and do not change to vesicles or pustules and are always associated with a thickening of the skin and marked itching. These characteristic eruptions have been found in the skin at the intermarginal part of the upper eyelid and the adjoining part of the conjunctiva.

Angioma of the Skin.—In conjunctival angiomata and teleangiectasias of the skin of the face, similar changes are found in the blood vessels of the lids and of the conjunctiva, and occasionally in the vessels of the retina and choroid.

Acne vulgaris of the face may extend to the eye lids, where the acne pustule is generally regarded as a stye.

Alopecia areata may affect the eye brows and the eyelashes, causing a partial or complete loss of hair.

Xanthelasma occurs frequently in the eyelids in the form of multiple flat or slightly prominent yellowish tumors which usually start at the internal angle, extending along the upper and the lower eyelids. They are often symmetrical, develop slowly and are permanent. Recently success has been obtained in the treatment of this condition with carbon-dioxide snow.

Burns of the Skin.—Retinal hemorrhages, optic neuritis and retinitis have been observed after burns of the skin. Apparently to produce these ocular changes it is not necessary that a large part of the body surface be affected. Wagenmann[1] believes that the eye lesions are the result of changes in the blood. Experiments in animals have shown that there is a destruction of red blood corpuscles which leads to hemoglobinæmia and multiple thrombi in the blood vessels are also an important factor. A similar process to that in the intestines and in the kidneys, occurs in the retina.

Seborrhœa affects the lid margin, causing it to appear red. The skin between the cilia is covered with small grayish scales which resemble the scales of the hairy scalp in dandruff. After removing these scales the underlying skin is found intact except that it is somewhat hyperemic. The cilia are normal and there are no ulcers, showing that the disease is not one of the cilia but of the sebaceous glands. Some authorities regard a seborrhœa of the lid margin as evidence of eye strain. It is more probably a parasitic disease which is influenced by a number of factors, particularly a disturbance of the general health, such as anæmia and scrofulosis. The condition is extremely chronic, though the symptoms can be very much improved and relieved by treatment.

Soft chancre is an unusual disease of the lid. Endlitz, mentioned by Michel, found in 66 cases of soft chancre that the eyelids were affected in 3 cases. The site of the lesion was usually the margin of the lid and the angle of the lids. The skin becomes red, an abscess forms in the layers of the skin. This breaks down and an ulcer forms with considerable purulent discharge. The ulcer has no particular characteristics and is of soft consistency. Accompanying this soft chancre there is an involvement of the preauricular gland. The ulcer sometimes progresses and involves the adjoining conjunctiva. After from two to four weeks cicatrization takes place and the scar remains soft.

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[1] Wagenmann, Arch. f. Ophth., Vol. XXXIV, p. 181, 1888.

Urticaria, a form of toxic dermatosis, frequently affects the eyelids where it produces a very striking amount of swelling and œdema, particularly of the upper lid, and closes the eyelids. The characteristic formation of blebs with itching is usually absent and occurs only when the skin of the rest of the face is involved.

XV. HEREDITARY EYE DISEASES[1]

The influence of heredity manifests itself when certain ocular conditions are met with in several members of different generations of the same family. Most hereditary eye diseases are directly transmitted from the father or mother to the children, without sex distinction. The inheritance is spoken of as indirect when it comes from a grandparent, uncle or aunt, thus skipping one or more generations. Collateral inheritance means the presence of a congenital lesion in several members of the same childship. A form of indirect inheritance by atavism is that in which the healthy daughter of an affected father transmitted the anomaly to her male children, a sex-limited condition; this is the rule in color blindness and congenital optic neuritis (Leber's disease). Another form of inherited disease is that occurring in the offspring of consanguineous marriages, such as retinitis pigmentosa. The inherited lesion occurs equally in the late as in the early-born children. Anticipation in hereditary disease means that the pathological condition tends to occur at an earlier age in the successive members affected. Nettleship speaks of it in connection with hereditary acquired cataract, hereditary glaucoma and in optic neuritis. An hereditary condition may sometimes invade different parts in different persons,[2] as in the occurrence of either retinitis pigmentosa, progressive nerve-deafness or feeble-mindedness as substitutes. A correlation seems to exist between Leber's disease and epilepsy; and albinism is often complicated with defects of the nervous system.

Certain diseases show a marked prevalence for a number of members of the same childship without much tendency to transmission to succeeding generations; these are called familial diseases.

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[1] Nettleship, Bowman Lecture, T. O. S., Vol. XIX, 1909.
Groenouw, Graefe Saemisch, II ed., Vol. XI, 2, p. 415, 1901.

[2] Nettleship, l. c., p. 74.

Lids.—Distichiasis has been observed as an hereditary lesion. The lids were normal, except for the presence of two rows of eyelashes; the extra row consists in very fine hairs situated at the inner lid margin.

Ptosis occurs as an inherited defect in one or more generations. The condition is congenital, sometimes without any other disturbance of motility though it is occasionally combined with epicanthus. Epicanthus has been observed alone (Manz).

Cornea.—Nodular and reticular opacity are allied conditions (Nettleship) which have been observed in from two to four generations of the same family, in other cases as examples of familial prevalence and in some single cases. Corneal staphyloma has been present in several members of the same family; as it is probably of inflammatory origin, the condition is not strictly inherited.

Iris.—Aniridia is a congenital anomaly which is frequently inherited. Parsons states that the influence of heredity is seen in this abnormality more than in any other ocular malformation. The degree of aniridia varies; even in the apparently total absence the condition is really incomplete. It is frequently accompanied by other ocular changes, such as corneal opacities, cataract, nystagmus and squint. The transmission is generally direct and both sexes are equally affected. Coloboma of the iris is another anomaly which is sometimes inherited, and is often associated with coloboma of the choroid. Corectopia or congenital excentric position of pupil is frequently inherited. With this anomaly the lens is sometimes subluxated, the lens and the pupil are then displaced in opposite directions. The condition is always bilateral and in addition to same being inherited, it occurs in several members of the childship.

Lens.—Cataract is not infrequently an inherited lesion; this does not mean a particular form of cataract, but a tendency to cataract. The inherited cataract in the affected members of a family is either always congenital or always acquired.

The mother on the other hand transmits the tendency to cataract to both sexes equally. The cataract may be transmitted through normal members of a family. If inherited cataract occurs in a family not all the members of a family are affected.

The form of cataract is not necessarily inherited; it is usually the one which is characteristic for the age at which the cataract occurs. Consanguinity of the parents is not frequent. As to the transmission, it seems, according to Groenouw,[1] that the father usually transmits the condition exclusively to one or a number of sons and sometimes to the grandchildren, but very rarely to any female child.

Nettleship has found that the discoid or "Coppock" form and the ordinary lamellar cataract are essentially the same. The discoid is probably only the smallest possible form of lamellar cataract, and its position at a deeper level than the normal nucleus is perhaps due to displacement backward of the nucleus from some developmental cause (Hess, Treacher Collins).

The usual-sized lamellar cataract is either congenital or acquired. Nettleship concludes that when lamellar cataract is hereditary, the small size of the lenticular opacity and the absence of dental deformity both point to the cataractous change having occurred during intrauterine life. The descent of lamellar cataract is always continuous. Hereditary influence is most strongly marked in the so-called coralliform[2] cataract in which the opacities extend forward from the central part of the lens, ending anteriorly in tubular expansions which are often intermingled with discrete spots of opacity. This form of opacity is sometimes included in the term, fusiform cataract. Of the acquired forms a number of examples are known of the transmission of senile, presenile and juvenile cataracts through several generations. The descent is in practically continuous passing through either sex, though preferably through the women. Hereditary cataract in one and the same family usually begins at the same age, though anticipation—the earlier incidence in each generation—occurs. It is stated that when senile cataract occurs as a family disease, it comes on unusually early. Some inherited cataracts, moreover, seem to lead to complete cataract formation (maturity) more rapidly than is usual.

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[1] Groenouw p. 436.

[2] Gunn, T. O. S., Vol. XV, p. 119.

Ectopia Lentis.—Congenital dislocation of the lens is frequently inherited. It has been observed in families for from two to four generations, and Morton has reported on a family in which this anomaly occurred in five successive generations. It is usually transmitted by the mother rather than by the father, and is distributed equally among the boys as well as the girls in these families. This congenital malposition of the lens is always bilateral and the lens is usually displaced upward in a symmetrical manner in the two eyes. The lens is generally clear and is stated to be smaller than normal. The condition is frequently associated with other anomalies of the eye such as coloboma of the iris, corectopia, abnormal diminution in size of the lens or of the entire eye ball.

Glaucoma.—Twenty-four families have been collected[1] in whom glaucoma was an inherited disease. The most striking features Nettleship finds in a strong tendency to anticipation in the younger generation and the continuous descent. Inherited glaucoma occurs at any time from puberty to old age. While glaucoma usually affects persons of advanced years, the inherited form is frequently observed in younger patients. In the same family the form of glaucoma does not vary; in other words, it is either inflammatory or chronic in all members.

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[1] Lawford, R. L. O. H., XVII, p. 57, 1907.

Buphthalmos, congenital glaucoma, is not an inherited condition but has occured in several members of a childship. This also applies to congenital microphthalmos.

Retinitis Pigmentosa.[1]—Primary pigment degeneration of the retina is a chronic disease of the retina which runs its course without inflammatory symptoms and with the characteristic pigmentation in the periphery of the eye ground. Nettleship[2] found proof of heredity in one-quarter of the families and of consanguinity of the parents or ancestors of those affected in another quarter. The descent of the disease is continuous from parent to child. The proportion of males to females is 3 to 2. Nettleship suggests that ill health after the acute exanthemata, tuberculosis, syphilis, or even severe loss of blood may bring out a liability to retinitis pigmentosa and that anything capable of damaging the arterioles might determine the onset of this disease in a predisposed choroid and retina. Retinitis pigmentosa may set in very early in life or even before birth; its onset is sometimes delayed until quite an advanced age.

The characteristic of the condition does not rest on the pigmentation, but in a progressive atrophy of the specific elements of the retina and a hypertrophy of the glia. The pigmentation is sometimes only slightly developed and may even be absent. This then gives rise to the term "pigment degeneration without pigment." *Retinitis pigmentosa sine pigmento* is always merely retinitis pigmentosa at an early stage before the pigment has become ophthalmoscopically visible; in rare cases pigment does not appear even in the late stages and the term is then appropriate. Leber suggests that these cases should be described under the term of "tapeto-retinal degeneration," of which there are two forms, the pigmented and the non-pigmented. A subdivision of the latter is the so-called retinitis punctata albescens, which term does not describe the character of the disease, but only a secondary complication which probably is due to an unusual development of colloid excrescences of the vitreous plate of the choroid.

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[1] Leber, Graefe-Saemisch-Hess, II ed., Vol. VII, p. 1076.

[2] Nettleship, R. L. O. H., XVII.

In retinitis pigmentosa the ophthalmoscopic picture is characterized by delicate fusiform or star-shaped pigment spots in the periphery of the eye grounds. The disc has a uniform yellowish-gray, wax-like color; the vessels are contracted. The retina frequently shows, especially in the upright image, a delicate veil which masks the drawings of the underlying choroidal stroma. The amount and distribution of pigment varies and the extreme periphery of the retina is usually free. The pigment spots are distinctly situated in the retina along the vessels. Nettleship has shown that the pigmentation appears only along the veins. The pigmentation is always found in the equatorial girdle and becomes lost in a direction forward as well as backward. A round area of typical pigmentation exceptionally occupies the macular region.

The visual disturbance consists in night-blindness and a slowly progressive ring-shaped later concentric contraction of the visual field, the central vision remaining preserved for a long time. Pronounced night-blindness is not always present. The central vision in the typical cases remains preserved for many years, though finally the process goes on to blindness. A frequent complication in the later stages is a posterior cortical cataract.

Cases in which the defect occupies a girdle have been repeatedly observed, and Gonin[1] was the first to show that the beginning of the disease always occurs in a zonular defect and that it is not until this defect extends toward the periphery that the concentric contraction of the field takes place, as is found in the later stages.

Gonin and Nettleship believe that the zonular form of field disturbance can be explained through the anatomic distribution of the choroidal arteries, and Hepburn[2] has attempted a classification of choroidal lesions according to vascular areas involved. Hancock,[1] however, has shown the position of the zonular defect does not correspond to the intermediate zone of the choroid where the two vascular layers are supposed to join, but it is in fact situated in a zone which is more posterior.

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[1] Gonin, Ann. d'oculist, CXXV, 1901.

[2] Hepburn, R. L. O. H., XVIII, 1910.

Pigment degeneration can occur at any time during life. Though it is considered to be congenital, its recognition early in life is difficult. Directly after birth pronounced retinal pigmentation or its preceding stage have never been found with the ophthalmoscope, and in the many examinations which have been made of the eyegrounds of the new-born, pigment degeneration has never been mentioned. In those cases in which retinal pigmentation has occurred in other members of the family, no signs of pigment have been found in the younger children. The ophthalmoscopic picture is often quite normal, even when there seems to be a defect in sight and where later on characteristic pigmentation takes place. In other cases there is a rarefication of pigment and the eyeground presents small bright dots; later on small pigment spots appear, followed by a deposit of pigment along the blood vessels.

The earliest symptom is the night-blindness, though this in some cases may be absent. There are cases in which the patients reach middle age before their sight becomes affected. Nettleship has observed cases that did not develop any symptoms before the fiftieth year. The usual course is progressive. In the thirtieth year the vision has definitely diminished. Between thirty-five and forty-five the patient is no longer able to follow his occupation and blindness practically occurs between the forty-fifth and the sixtieth year. The remaining central vision is lost by the development of a posterior cortical cataract. This cataract remains limited to the posterior cortex and only rarely becomes complete. Complete blindness does not necessarily occur in all cases.

A remarkable complication is secondary glaucoma, which is so frequent that an accidental association seems improbable. The most frequent extraocular complication is deafness, and statistics have been made on the frequency of pigment degeneration in deaf-mutes and in idiots.

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[1] Hancock, R. L. O. H., XVI, 1906.

Some observers regard cases of unquestionable syphilitic origin as examples of pigment degeneration of the retina, while they should be more properly called chorio-retinitis with secondary retinal pigmentation. The simple pigment degeneration usually occurs without choroiditis, while the syphilitic variety appears as a chorio-retinitis. The disease of the choroid, with its pronounced inflammatory character, particularly shown by the presence of multiple circumscribed exudates and vitreous opacities, are characteristic for the latter condition. Furthermore, the course is more subacute and varies in its progress. The pigment spots are also different. Leber believes, however, that syphilitic retinitis may present the typical ophthalmoscopic picture of a pigment degeneration, not only in the acquired but in the inherited form.

Among the congenital cases there are some which remain stationary. The acquired retinitis pigmentosa, on the other hand, is generally progressive.

Among the inherited cases the various forms may all be represented in the same family. The acquired retinitis pigmentosa develops from the fifteenth to the twentieth year. In these cases heredity is at fault. In the cases which develop later there is no inherited factor.

In addition to the retinitis pigmentosa these cases often are deaf-mutes, or suffer from deafness, feeble-mindedness, general development defects, such as supernumerary fingers and toes. Retinitis pigmentosa seldom affects both parents and children; it is seen in children of healthy parents where the parents are related, and the closer the relationship of the parents the more serious is the inherited lesion in the children. The affected members of a family usually show about the same degree of disturbance and the amount of pigment can be taken as a guide for the duration of the disease.

As has been stated, pigmentary retinitis will alternate with other ocular diseases in the same family. In other words, it has been observed to occur accompanied with atrophy of the optic nerve, strabismus, nystagmus and excessive mortality among the children is frequently observed in these cases. In some cases the disease apparently seems to occur in every other child, the intervening child having normal eyes.

Retinitis pigmentosa does not hinder fertility; the families in which this disease occurs are often large. It is not possible to speak of the relative frequency of retinitis pigmentosa, of deafness and of feeble-mindedness in the same pedigree. Nettleship says that certain stocks produce only retinitis pigmentosa while others only the equivalent deafness.

The So-called Pigment Degeneration Without Pigment.[1] —In the beginning of retinitis pigmentosa retinal pigmentation is not present, and the pigment epithelium only shows finely dotted rarefication and atrophy. Pigmentation may not become visible to the ophthalmoscope for a long time, even in the stage in which the disc assumes the the characteristic yellow, wax-like appearance and the vessels have become noticeably contracted. The ophthalmoscopic picture is characteristic even without the presence of pigmentation, and the diagnosis is readily made, especially when night-blindness is present.

The so-called pigment degeneration without pigment may exist for many years, if not during the entire life of the patient. The importance of this variation was first insisted upon by Leber in 1871; a recent paper on the subject is by Gebb.[2] The association with pigment degeneration is clear from its familial and inherited appearance, also from the fact that in the same family there will be cases without

[1] Leber, l. c., p. 1174.

[2] Gebb, Arch. f. Augenh'lk., Vol. LXIX, 2, 1909.

pigment, and others with pigment to a varying degree. Leber speaks of a diffuse, whitish-gray opacity in some cases where the pigment is lacking, which seems to be situated directly in front of the pigment epithelium, or in the deeper layers of the retina. These cases are often regarded as examples of hemeralopia, but they belong to the "tapeto-retinal" degeneration because, in addition to the ophthalmoscopic condition, there is night-blindness, contraction of the field and some retinal pigmentation. A careful description of the various stages of the process has recently been made by Lindner.[1] The opacity may extend quite a distance over the eyeground and gives it a peculiar appearance. The disc is sharply outlined and the vessels pass unobstructedly. The opacity is diffuse and in the early stages at its periphery breaks up into streaks and spots which have a net-like arrangement. The macula is free but is surrounded by a zone of radiating streaks or points. The process shows a continuous change and the dots gradually disappear. In addition to the other symptoms, a central scotoma is present. There was consanguinity of the parents.

Retinitis punctata albescens[2] is a form of the non-pigmented tapeto-retinal degeneration (Leber) in which the eyeground is covered by numerous small sharply defined white points or dots. Otherwise the eyeground shows the signs of the usual non-pigmented pigment degeneration with some variations belonging to the pigment form, especially as regards the functional disturbance.

Leber is of the opinion that this is a variety of the non-pigmented form in which the presence of the white spots is an unessential variant. These changes are always stationary and are situated in the external layers of the retina or on the internal surface of the choroid. The white spots are probably well-developed and partially calcified colloid excrescences of the vitreous plate. They are usually round, sharply defined, discrete or collected in small groups.

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[1] Lindner, Graefe's Archiv., Vol. LXXXVIII, 2, 1914.

[2] Leber, l. c., p. 1177.

The disturbance of sight is of varying grades: there is night-blindness, and central vision may or may not be affected. The field is concentrically contracted. In many cases the condition is stationary and the visual disturbance is then limited to night-blindness.

Nettleship[1] has brought forth a number of reasons why the spots are not colloid excrescences. Aside from the characteristic disturbance of vision, the association of the so-called retinitis punctata with the pigment degeneration is shown in its familial appearance and the frequent consanguinity of the parents.

Pigment Degeneration of the Retina with Extensive Choroidal Atrophy (Atrophia gyrata choroideæ et retinæ).[2] —Retinitis pigmentosa is sometimes associated with extensive atrophy of the choroid and the eyeground shows a very striking shiny white appearance.

There are more or less typical disturbance of vision, pigmentation of the retina with extensive atrophy of the pigment epithelium, and of the choroid. Fuchs and Cutler described cases where the choroidal atrophy was only partial, under the name of atrophia gyrata.

The association with the retinitis pigmentosa group is clear from the characteristic form of the visual disturbance, the retinal pigmentation, its familial appearance and the parental consanguinity. Posterior cortical cataract is also present. Leber believes that the changes are due to some pathological process which terminates very early in life. In the atrophia gyrata there are peripheric atrophic areas with many pigment deposits and sharply limited toward the disc with an irregular margin. In the extreme periphery the fundus may be normal and it would seem that the disc was surrounded by an atrophic girdle. In some cases the atrophy is limited to the pigment epithelium and the choroid is not very much changed.

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[1] Nettleship, R. L. O. H., XVII, p. 377, 1908.

[2] Leber, l. c., p. 1185.

Amaurosis from Tapeto-retinal Degeneration.[1]—There is a form of retinal degeneration belonging to the retinitis pigmentosa group which rapidly leads to complete blindness. Leber calls it "amaurosis from tapeto-retinal degeneration." The cases occur in two groups: infantile, where the blindness is observed in the first year; and juvenile, in which it appears between the end of the first year and the onset of puberty.

In a part of these cases there is an associated cerebral degeneration which may lead to complete imbecility, resembling the so-called amaurotic family idiocy with which they are, however, not identical. The tendency of this disease to occur in a familial manner is striking except in the uncomplicated infantile form.

According to Leber, the infantile form included most of the cases of congenital or early acquired blindness from affections of the retina or of the optic nerve. Congenital affections of the optic nerve are much more uncommon. The family amaurotic idiocy is also much more uncommon. It is noted that these children do not grasp for objects and the eyes are not steady; nystagmus is usually present. The pupils are moderately wide and there is slight reaction to light.

In older children the vision seems to vary between complete loss of light perception and more or less amblyopia without night-blindness. The pronounced nystagmus excludes a determination of the field. In the first months of life the ophthalmoscopic examination shows no striking change. In the second half of the first year the pigment epithelium in the periphery and sometimes near the disc shows the characteristic changes of the typical pigment degeneration, namely, a finely granular condition consisting of small closely grouped bright dots with intervening pigment points. The retinal vessels are somewhat narrow, the disc shows no change. Fine pigment points are distinctly present at the age of two and one-half years, when the typical pigmentation along the blood vessels begins to occur. The pigmentation in these cases gradually increases; and the disc turns grayish-yellow.

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[1] Leber, l. c., p. 1188.

Another group is made up by the cases in which the disease does not develop until later in life, between the fifth and ninth year, and which Leber designated as the juvenile form. The cases of Stock[1] and Spielmeyer,[2] in which there was an associated rapid loss of mental faculties, belong to this group. This form is to be separated from the cases of pigment degeneration and congenital idiocy. There are other cases which belong to this group, in which the blindness and the dementia occurred in the first years of life, so that we can distinguish two forms of both the infantile and the juvenile varieties, the simple form and the one complicated with dementia.

The simple juvenile form is very unusual. The form complicated with the acquired dementia resembles the amaurotic family idiocy, though it is different from this disease.

Batten[3] and Mayou[4] have described a familial disease consisting in tapeto-retinal degeneration localized to the macula associated with cerebral degeneration similar to the one observed in the previous cases. In these cases the disease occurred between the fifth and the ninth year, and attacked a number of children in the same family. Its association to the pigmentary degeneration group is shown by the changes in the pigment epithelium which are scattered over the eyegrounds, the contraction of the retinal vessels, though the pronounced changes in the macular region are an unusual complication.

As to the etiology, the same factors are present which occur in the typical pigment degeneration, except that direct inheritance has not been observed while consanguinity and its appearance in families corresponds to the pigment degeneration.

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[1] Stock, Kl. M. A., XLVI, 1908.

[2] Spielmeyer, Centralbl. prakt. Augen., 1905.

[3] Batten, T. O. S., Vol. XXIII, 1903.

[4] Mayou, T. O. S., Vol. XXIV, 1904.

A striking complication is the history of severe convulsions. In these cases, aside from the association with idiocy, nervous symptoms are very much more frequently present than in the ordinary pigment degeneration.

Familial Tapeto-retinal Degeneration of the Macula and Disc Regions.[1]—There is an unusual disease of familial or hereditary appearance where the pathological changes are limited to the macula and disc regions. In addition to the influence of heredity, this condition, according to Leber, belongs to the group of "tapeto-retinal" degeneration and there may be a pronounced development of colloid bodies. The condition is usually referred to as chorioretinal degeneration, but there is no pigmentation of the retina.

The localization to the posterior pole explains the amblyopia, a central scotoma instead of a ring scotoma or a peripheric contraction of the field. Hemeralopia is usually absent.

Another point of similarity with other forms of tapetoretinal degeneration lies in the association with cerebral degeneration leading to imbecility.

The ophthalmoscopic changes consist in some cases in the presence of small pale yellow or gray spots scattered over the region of the macula and the disc. In other cases there are circumscribed areas of atrophy and irregularity of the pigment epithelium, or there may be black pigment deposits of varying size or shape. In some cases bright spots have been noted which resembled colloid excrescences. Hutchinson and Tay[2] described a condition somewhat resembling this in three sisters, and called the disease "guttate choroiditis." Except for the bright spots in the region of the macula and disc, the eye ground is normal. Doyne[1] reported upon an ophthalmoscopic condition which he found in several members of a family where the white spots in this region were much larger and were grouped in a honeycomb arrangement. Oatman[2] has reported on a family presenting the condition just described, and gives it the name of "family maculo-cerebral degeneration," in distinction from the cases of uncomplicated macular lesion.

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[1] Leber, l. c., p. 1204.

[2] Quoted from Leber, p. 1205.

The disease is nearly always bilateral. It occurs at various ages, generally not before the tenth to the fourteenth year. The disease is usually limited to several siblings though heredity and consanguinity have been noted in some cases.

Hereditary night-blindness is an unusual condition in which night-blindness is present from birth without ophthalmoscopic changes. In daylight the field of vision is not restricted. The condition is stationary and is not associated with any other defect. It is not possible to say whether the same changes are present which occur in pigmentary degeneration of the retina, except that these two conditions are sometimes observed in the various members of the same family. The disease, however, is more frequently inherited and occurs in a number of brothers and sisters, while the consanguinity of the parents is less frequent. Nettleship distinguishes between two groups. In one, both sexes are equally affected, with continuous descent through either parent without any other peculiarity of the eyes nor any change in the fundus. In the other group the condition is limited to males with descent through normal-sighted females and with myopic refraction. The vision with correction is often subnormal. Slight changes are sometimes found in the fundus and nystagmus has been noticed in a few.

Hereditary Optic Neuritis.—The first complete description of this remarkable clinical picture was given by Leber.

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[1] Doyne, T. O. S., 1899.

[2] Oatman, Am. J. Med. Sc., 1911.

The disease develops as a retrobulbar neuritis usually a few years after the onset of puberty, around the twentieth year. This condition is usually symmetrical and simultaneous in onset, though there have been cases described in which the intensity in the two eyes has differed, leaving one eye untouched. As a rule, an interval of a few weeks between the onset of one eye after the involvement of the other is not infrequent. The condition usually begins quite abruptly, reaching its acme after a few weeks or months, with no further change taking place, and a central scotoma remains as a permanent feature. The periphery of the field is usually intact. Though total blindness has been described in some cases, Nettleship is of the opinion that this rests upon incomplete data.

The ophthalmoscope reveals at the beginning slight changes such as a clouding of the margins of the disc and in some cases the eye grounds seem to be perfectly normal. After a number of months the optic nerve shows a distinct pallor which is either complete or limited to the temporal quadrant. As a rule, the central scotoma is a permanent defect. A restoration, complete or in part, has also been observed. Nettleship believes that the chances of recovery have been put too low, and it is not necessary to speak without hope of any case seen within a couple of years from its onset. He reports 25 cases in which 16 geneologies recovered either completely or useful central vision, while minor improvements are quite common. A very important feature is that the time before noticeable improvement takes place may be quite long, often twelve to eighteen months.

The various members of the same family usually run the same course. The true cause for this remarkable condition which consists in a selective invasion of the papillomacular bundle is not known. Though the disease usually starts around the twenties, it has been observed earlier, at five, and as late as the sixty-seventh year. A number of cases have been observed in which the condition develops between the forty-first and forty-ninth year. The characteristics of the disease are the same at whatever age the attack occurs. In the families in which the disease comes on before the fourteenth year some females are usually affected, while in those in which it is deferred until thirty or later, female cases are very unusual. Aside from the early deaths, there is no particular prevalent morbid tendency in these families. The most frequent are epilepsy and aggravated hysteria. The victims are usually males, while the descent takes place through the unaffected mother. Consanguinity of the parents is very unusual. In a few cases there has been a history of other neuroses (usually epilepsy) in the patient or his relatives.

Nettleship has found 60 females affected, as compared with 300 males. The age of onset is usually the same for all cases in the same geneology. There is a tendency to anticipation in this condition, though it is not so pronounced a symptom as in glaucoma or senile cataract.

The number of children born in families containing cases of this disease is not less than normal though Nettleship has observed that if there are many births the early mortality is greater than normal among the male children.

The scarcity of consanguinity in Leber's disease is what is to be expected in a sex-limited affection. The diagnosis may be difficult when it is necessary to distinguish this condition from familial optic atrophy due to tower skull or other cranial deformities. There is no connection between Leber's disease and tapeto-retinal degeneration with macular changes.

Hereditary Nystagmus.—Nettleship has found 25 pedigrees of hereditary nystagmus in the literature to which he has added a few additional ones. Infantile nystagmus when it occurs as a family affection and not due to a primary nervous cause, is a symptom of defective sight. This defect in sight dates from birth, often presenting no ophthalmoscopic changes. It sometimes appears inherited in a number of generations in the same family, with weaksightedness due to a high grade of hypermetropia and astigmatism, though poor sight has been observed in cases without these errors of refraction. In some families the male members have been particularly affected.

It seems that nystagmus is more easily produced in some persons than in others, which may explain the cases in which there is only slight defect in vision or only a moderate degree of astigmatism. Generally the defect in sight is considerable. Nystagmus of this kind sometimes becomes less marked in later years, and it has been known to cease. It is sometimes less marked in some one position, which position varies with the individual.

Nystagmus is an important symptom in hereditary ataxia, a condition which usually affects several members of a family. The inheritance is rarely direct and consanguinity in the parents is frequent. A general neuropathic failing is common, showing itself in other nervous or mental diseases.

Aside from the very early defects in the eyegrounds which run in families and cause hereditary nystagmus, there are two conditions which are chiefly responsible for hereditary nystagmus, albinism and total color-blindness.

Albinism.—In this condition the pedigrees occurred with and without consanguinity, the descent is either continuous or discontinuous, and is sometimes associated with correlated diseases. The iris is usually of a blue or gray color, the hair brown, although very white in early childhood, with a more or less albinotic eyeground, nystagmus and marked amblyopia. With increasing age the vision is sometimes improved and the photophobia is diminished. Albinos, as is generally known, are weak individuals who usually die young, before the fortieth year. In the cases in which the sight is good and the eyes steady, Nettleship is inclined to assume that the retinal epithelium, particularly in the region of the yellow spot, is lacking pigment. There are all degrees of albinism. In the partial cases the condition is almost perfectly sexlimited. Of 43 affected persons, 40 were males and the descent was through the mother in every case. No affected male, however, had an affected child. In general albinism the descent is usually discontinuous. Consanguinity of the parents is common. All the members of a childship do not suffer from albinism.

Color-Blindness.—Congenital color-blindness is either total or partial. The total color-blind person has no appreciation for color. Everything is gray; colors simply mean differences in luminosity. The vision under these circumstances is very much reduced. Nystagmus is a frequent symptom. The patients say that they see better in a dull light and during the daytime they claim to be blind, a condition which resembles toxic amblyopia. The disease often occurs in a number of members of the same childship. Nettleship states that it does not occur in both parent and child. Eighty-four cases have been thus far collected. Mental defects are relatively frequent. In its general characteristics this condition suggests typical pigmentation of the retina.

Partial color-blindness, on the other hand, is usually not associated with other defects in the eye, and vision is usually good. Investigations have shown that partial color-blindness occurs in males in about 3 per cent. and in females in about 0.3 per cent. The most frequent form is red-green blindness. This form of color-blindness occurs not infrequently in a number of members of the same family. As men are so much more frequently affected than women, it will be generally observed in brothers. The form of inheritance is the usual one in sex-limited anomalies, where the female members of the afflicted family are not affected but transmit the trouble to their sons. Color-blindness exceptionally is transmitted by the male members of a family. Consanguinity of the parents is unusual.

Defects in motility of the eye are sometimes congenital. They vary in degree from a slight ptosis to a paralysis of all the external muscles. In the pronounced cases the ptosis is bilateral and there is a more or less complete paralysis of all of the external muscles. A paralysis of the associated lateral movements has been observed with preserved convergence. The pupil and the accommodation are always intact, but the vision is frequently reduced. The cause is not a uniform one. In the congenital ptosis the cause seems to be a change in the muscle itself as the nerve and the nucleus are not affected. Congenital ptosis occurs alone or with other anomalies. Ptosis has been observed in a number of successive generations and the transmission is usually direct. It is sometimes associated with epicanthus and blepharophimosis.

According to Groenouw, in the families in whom other defects of motility aside from ptosis occurred, the form of inheritance was collateral or direct in two to three generations. The defect was the same in kind, though differed in degree in the members of the same family.

Errors of Refraction.—The question of the inheritance of errors of refraction is a difficult one to solve. The result of a great deal of investigation on the heredity of near-sightedness has shown that a certain definite percentage (30 per cent.) of myopes show a distinct inherited tendency which has favored the development of near-sightedness. The near-sightedness apparently then develops under unfavorable conditions, especially in patients who have an hereditary tendency. This inherited disposition to near-sightedness consists in a diminished resistance of the sclera by which the eye ball becomes elongated through the agencies which are active. Inherited variations in the structure of the ocular muscles and in the shape of the orbit may be of importance in this connection. The influence of heredity becomes more marked as the degree of the myopia increases. The descent is nearly always direct and the influence of the father is greater than that of the mother and the influence is exerted about equally on both the sons and daughters. Investigations on other forms of refractive errors have not been made to the same extent. There can, however, be no question that astigmatism both in its form and in the position of its axes, is an inherited evil.

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